THE IMPACT OF MEDICARE ON LIFE EXPECTANCY*

Andrew Goodman-Bacon

Adriana Lleras-Muney

Joseph Price

Dahai Yue

October 23, 2025

Abstract

This paper estimates the causal effects of Medicare on mortality rates and life expectancy among the program's early recipients. We construct a new dataset of more than 18 million individuals observed in the 1940 census linked to a death record in the FamilyTree database at FamilySearch. We use Medicare's introduction in 1966 to identify its average treatment effects using three pre-specified approaches: a design based on a simple theoretical model of cohort mortality, an interrupted time-series design, and a staggered difference-in-differences design. All three show that Medicare increases life expectancy at age 65 for men born between 1885 and 1915 by an average of one year. Medicare's effects on life expectancy at age 65 are larger for cohorts with more potential years of exposure but similar for groups of high and low socio-economic status. The effects for women are not robust across methods and specifications. *JEL Codes*: H51, I13, I18.

---

*Research reported in this publication was supported by the National Institute on Aging of the National Institutes of Health under Award Number R21AG080416. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. Adriana Lleras-Muney benefited from facilities and resources provided by the California Center for Population Research at UCLA (CCPR), which receives core support (P2C-HD041022) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD). The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health & Human Development or the National Institutes of Health. We are grateful to Charles Hutchinson for outstanding research assistance, and to Julian Reif, Maurizio Mazzocco, and the seminar participants at the Population Association of America 2025 Annual Meeting, 2025 ASHEcon Conference, Princeton University, UC San Diego, Emory University, University of Essen-Duisburg, Technical University of Dortmund, University of Cologne, the NBER Aging group, the NBER health group, the Federal Reserve Bank of Minneapolis, and University of Queensland for their valuable comments.

Corresponding author: Andrew Goodman-Bacon, 90 Hennepin Ave, Minneapolis, Minnesota 55401.

I. INTRODUCTION

In the United States, Medicare, a federal health insurance program, is the primary source of coverage for people aged 65 and older. In 2023, Medicare enrolled 65.1 million people, cost over one trillion dollars, and accounted for 21 percent of US health expenditures and 3.7 percent of Gross Domestic Product (Keehan et al. 2025). Almost everyone older than 65 is enrolled (Keehan et al. 2025; Moon 1996), and 91 percent of them rate Medicare's performance highly (Pollitz et al. 2023). As a result, Medicare has been put forth as a blueprint for extending health insurance to the entire US population. But because Medicare's costs have also ballooned, many have also proposed reforms to reduce its size.

Alongside providing financial protection, which it has successfully done since its inception (Barcellos and Jacobson 2015; Caswell and Goddeeris 2020; Finkelstein and McKnight 2008; Goldsmith-Pinkham, Pinkovskiy, and Wallace 2023), Medicare aims to maintain population health by providing access to basic health care. An important justification for Medicare spending thus hinges on how well it supports medical care consumption among older people and the efficacy of that care in maintaining and improving their health.

Yet, estimating causal effects of Medicare on the health and mortality of elderly recipients has proved difficult. One reason is that Medicare provides universal coverage at age 65 and has changed only a handful of times in its history. Studies focused on the age-65 discontinuity find that one-year mortality falls sharply at age 65 among people with urgent hospital admissions (Card, Dobkin, and Maestas 2009), but not in the overall population at age 65 (Card, Dobkin, and Maestas 2004; McWilliams et al. 2004; Polsky et al. 2009). Studies of Medicare's introduction in 1966 (Chay, Kim, and Swaminathan 2010; Finkelstein and McKnight 2008), its extension to people with end-stage renal disease in 1972 (Andersen 2018), and its coverage of prescription drugs in 2006 (Huh and Reif 2017) find mixed effects on mortality.[1]

Another challenge is that none of these studies observes longer-term mortality rates, when Medicare's effects may be largest. Theoretical models (Dalgaard and Strulik 2014; Grossman 1972; Lleras-Muney and Moreau 2022) and empirical studies (Leinonen, Heikkinen, and Jylhä 2001; Levinsky and Schiff 2021) document that health accumulates and deteriorates slowly. Therefore, the benefits of Medicare, which increases the use of preventive care, chronic care

[1] Estimated effects of public health insurance for low-income non-elderly adults have this pattern, too. Graves et al. (2020), Miller, Johnson and Wherry (2021), and Wyse and Meyer (2025) all use specific data on low-income mortality rates and find health improvements that are indetectable in aggregate data (Black et al. 2022).

management, and diagnostic tools (Card, Dobkin, and Maestas 2008; Finkelstein 2007; McWilliams et al. 2007; McWilliams et al. 2003), may not materialize for many years, especially among relatively healthy people. Indeed, recent evidence suggests that providing health insurance to low-income children has significant long-term impacts (Boudreaux, Golberstein, and McAlpine 2016; Brown, Kowalski, and Lurie 2020; Goodman-Bacon 2021; Miller and Wherry 2019; Thompson 2017; Wherry and Meyer 2016; Wherry et al. 2018). Whether this evidence applies to adults who are no longer growing is unknown.

This paper estimates the causal effects of the introduction of Medicare on the mortality rates and life expectancy of the program's early recipients. Medicare began providing health insurance for hospital stays and physician visits to (almost) all Americans over the age of 65 in July 1966. Only about half of this group had health insurance in the early 1960s; they faced large medical costs, and often forwent potentially life-saving medical care (CHAS and NORC 1984). Soon after Medicare's passage, however, people over 65 began to use substantially more medical care and faced lower financial risk (Finkelstein and McKnight 2008).

We generate hypotheses about how the existence of Medicare may have affected its recipients using the theoretical model of cohort mortality in Lleras-Muney and Moreau (2022). This framework first predicts that Medicare should have dynamic effects on a cohort's log mortality rates that vary with the amount of time they have been eligible. Additionally, the shape of these effects provides evidence on whether Medicare changed the evolution of health (investments or depreciation rates) or the severity of health insults (the variance of health shocks or the death threshold). These insights suggest that the key quantities necessary to understand Medicare's effects on longevity are causal effects on mortality for a given cohort in the years after they become eligible for Medicare. Our empirical analysis targets these parameters.

Our ability to estimate such effects hinges on detailed measures of cohort mortality based on a new dataset of more than 18 million white people observed in the 1940 census who can be linked to a death record. The age profiles of mortality in our data closely match those from both the Social Security Administration and the Human Mortality Database. However, we also observe covariates that enable us to investigate *heterogeneity* in ways that are not possible using existing data sources. These data allow us to map causal effects on mortality rates into a *long-run* outcome: cohort life expectancy conditional on surviving to age 65.

We pre-specify three complementary research designs that identify these effects under distinct assumptions.[2] First, for each cohort, we estimate the parameters of the theoretical model using pre-Medicare data and use them to construct counterfactual mortality rates without Medicare during their post-Medicare years. The validity of this approach depends on the correct specification of the model and the reliability of its parameter estimates. Our second approach is an interrupted time series (ITS) design based on the assumption that untreated log mortality rates in adulthood are linear in age, which allows us to form counterfactual mortality using the estimated pre-Medicare slope of a cohort's log mortality age profile. Linearity is a qualitative prediction of the model and a well-known empirical regularity first observed by Gompertz in 1825 (Gompertz 1825). Finally, we employ a staggered difference-in-differences (DiD) design that compares changes in log mortality across cohorts. Here, we rely on a parallel trends assumption that justifies using observed mortality changes for older cohorts who gained Medicare later in life to form counterfactual mortality trends. All three designs identify the impact of Medicare as it was experienced by affected cohorts between 1966 and 2015, on average, 14-18 years later.

Results from all three empirical approaches align closely for men, showing that Medicare increases life expectancy at age 65 by about one year on average: the effect gradually increases from zero to roughly 2 years across the 1885-1910 birth cohorts and then decreases for the 1915 birth cohort. Increases in life expectancy arise from dynamic effects on log mortality rates that are initially small but grow with time on the program. These findings closely match the predictions from our theoretical model and suggest that Medicare either increases health investments or slows the rate of aging. We also find that Medicare had somewhat greater effects on the log of mortality for lower-educated subgroups, but these effects do not translate to detectable differences in life expectancy except for women with the least education. The results for women, however, are inconclusive. DiD produces results similar to those for men, but ITS results often has the opposite sign.

Ours are the first estimates of the effect of Medicare on long term mortality and life expectancy. Previous work, such as Finkelstein and McKnight (2008)'s seminal study of Medicare's introduction, necessarily relies on coarser age groups available in Vital Statistics data. Our data provide accurate mortality rates by single year of age, which allow us to implement new

[2] To empirically ground our design choices and still conduct valid statistical inference, we use a random 20 percent of our sample to test identifying assumptions and make key specification choices. Our full analysis plan and code will be archived with the Open Science Framework.

identification approaches that uncover meaningful long-term mortality reductions. The program's introduction is also the only opportunity to observe large groups who first became eligible for Medicare at ages older than 65, and we find very similar mortality effects across these groups on their log mortality rates. In principle, our estimates also incorporate spillover effects, although we find little direct evidence of them, despite Medicare's documented spillovers onto the healthcare sector (Clemens and Olsen 2021; Finkelstein 2007). Medicare has grown tremendously in scope, scale, and cost since 1966. Our findings suggest that these outlays likely still confer meaningful benefits to population health.

Our findings also add to a growing number of reevaluations of the health effects of President Lyndon Johnson's Great Society programs. Like us, this work documents substantial long-run benefits of Medicaid (Boudreaux, Golberstein, and McAlpine 2016; Clay et al. 2024; Clayton 2019; Goodman-Bacon 2018; Goodman-Bacon 2021), Head Start (Ludwig and Miller 2007), food assistance (Hoynes, Schanzenbach, and Almond 2016) and community health centers (Bailey and Goodman-Bacon 2015). A key lesson is that carefully designed studies with long time frames frequently uncover strong evidence of benefits that short-run correlational evidence misses (see also Aizer et al. 2024; Aizer, Hoynes, and Lleras-Muney 2022).[3]

II. INSURANCE AND HEALTH CARE BEFORE AND AFTER MEDICARE

Poor health was an increasingly burdensome problem for American seniors during the decades prior to Medicare's passage. Private health insurance, initially developed in the 1920s, exploded after World War II (Thomasson 2003). The war also gave millions of soldiers new experience with regular medical care, and their subsequent demand for it was met with a rising supply. Innovations developed during WWII, such as antibiotics, blood transfusions, and ultrasounds (e.g. Van Tiggelen and Pouders 2003) found civilian applications, and new federal hospital subsidies greatly expanded capacity (Chung, Gaynor, and Richards-Shubik 2017). These developments were good for population health, but created an intractable affordability problem for seniors.

People over age 65 got little financial protection from private insurance. Since many were retired, they rarely had insurance through employers. Individual market policies were medically underwritten and thus unavailable or prohibitively expensive. Blue Cross Blue

[3] We are not aware of other papers that show longer-run effects for elderly people, except for Deryugina and Reif (2023) who use the model in Lleras-Muney and Moreau (2022) to demonstrate that the long-term effects of pollution on mortality are larger than short term effects.

Shield sold community-rated plans, but adverse selection pushed up their premiums as well (Thomasson 2004). **Figure 1a** plots the age profile of health insurance rates for white respondents in the 1963 National Health Interview Survey (NHIS) and shows that just 56 percent of people age 65 and older had any health insurance coverage. Moreover, insured people still faced substantial out-of-pocket costs. Most only held a policy that covered hospital expenses, and even those policies were far from comprehensive. Just over half of elderly insured people with hospital stays in 1962 reported that insurance paid the majority of their bills (Epstein and Murray 1967, Table 11.13).

Without the financial protection of insurance, "[c]hoices for uninsured or underinsured elderly patients needing hospital service were to spend their savings, rely on funding from their children, seek welfare…or hope for charity from hospitals" (Stevens 1996). A small number of low-income elderly people had their care financed by welfare departments or lived in state institutions.[4] Charity providers often provided low quality care, had long wait times, and could refuse service or pursue aggressive collection practices (Stevens 1989).[5]

Statistics on the use of basic health care services reflect these problems. **Figure 2b** plots the age profile of the number of nights in hospital in the past 12 months over age pooling data from 1963-1965 NHIS. Prior to 1966, the data show small gaps in the intensity of hospital use for older versus younger people; a sharp contrast with the measurably poorer health of the elderly.[6]

In response to these conditions, the Johnson administration made Medicare the signature component of the 1965 Social Security Act amendments.[7] Medicare provided two kinds of

[4] These programs included Old Age Assistance, which paid for some medical costs since 1950, or the short-lived Medical Assistance for the Aged program, which mainly operated in a handful of large states. These programs were small. They means test, their legal claim on the resources of a recipient's children made many people reluctant to apply, and they often did not cover important services (Goodman-Bacon and Nikpay 2017).

[5] In 1959, a Senate subcommittee on aging held hearings during deliberations on a health insurance bill introduced by Senator Aime Forand. One staffer reported that "The old folks lined up by the dozen every place we went…and they didn't talk much about housing or recreational centers or part time work. They talked about medical care" (quoted in Starr 1982, page. 368).

[6] Relative to people aged 50-64, respondents aged 65-79 in one 1963 survey reported more serious symptoms like shortness of breath after light work (29 versus 16 percent), sudden weakness or faintness (26 versus 17 percent), or painful/swollen joints (42 versus 31 percent) (CHAS and NORC, 1984). Mortality rates in 1960 were more than three times higher for the 65-79 group than the 50-64 group (4,557 versus 1,433 deaths per 100,000), and more of these deaths came from cardiovascular causes (49 percent versus 41 percent) (NCHS, 1963).

[7] Despite the long history of public health insurance bills, "the outcome of the debate over Medicare was by no means predictable" (Stevens 1989; pg. 47). A Medicare bill pushed by the Kennedy administration was narrowly defeated in 1962, but during the 1964 presidential campaign President Johnson "repeatedly promised that if her was elected he would see to it that Congress enacted a program of national health insurance for men and women sixty-five and over" and after the Democrats gained control of congress "no one in the government doubted that he would get it" (Harris 1966). The administration proposed a universal hospital insurance program for the elderly with no premiums and minimal cost-sharing. Congressional Republicans preferred a means-tested system run by

insurance to almost all people age 65 and older. Part A provided compulsory hospital insurance with a $40 annual deductible and no cost sharing for stays under 60 days. Part B offered optional medical and surgical insurance (without drug or long-term care coverage) with 20 percent coinsurance and a $3 per month premium (Gornick et al. 1996). 93% of seniors enrolled in it (West 1971). Upon signing it into law, President Johnson declared, "no longer will older Americans be denied the healing miracle of modern medicine" (Peters and Woolley 1999).

Medicare officially began on July 1, 1966, and it immediately and dramatically increased insurance coverage for the elderly. **Figure 1** also plots the age profile of health insurance coverage from the 1968 NHIS and shows a large jump at age 65. Health insurance rates rose for all ages, but for people aged 45-64 the average increase was 5 percentage points, while for people over 65, the average increase was 41 percentage points. Given the incompleteness of insurance coverage, Finkelstein (2007) estimates an even larger 75 percentage point increase in "meaningful health insurance." One survey of Social Security recipients found that in Medicare's first year, patients paid about 47 percent of medical costs compared to 77 percent in the year prior (West 1971). Using an age-based DiD design, Finkelstein and McKnight (2008) find that Medicare cut out-of-pocket health care spending in the top decile in half.

Changes in health care use reflect these dramatic changes in health insurance coverage. **Figure 1b** shows that the average annual number of nights spent in a hospital among people over 65 jumped from about 2 to 3 after 1966, while it stayed flat for people between the ages of 55 and 64. Today, this remains true: Individuals on Medicare use health care at much higher rates than those without (Card, Dobkin, and Maestas 2008), and they have lower medical expenditure risk (Barcellos and Jacobson 2015).

the states, and the American Medical Association proposed subsidizing the purchase of private fee-for-service insurance plans. Ultimately, the legislation that created Medicare combined all three proposals. Even this was a surprise: "On March 12, 1965 [Senate Finance Committee Chair, Wilbur] Mills stunned [Undersecretary of the Department of Health, Education and Welfare, Wilbur] Cohen by asking if it would be possible to amalgamate a compulsory hospitalization program, a voluntary medical insurance program, and an expansion of coverage for the indigent" (Cunningham and Cunningham 1997; pg 143).

III. POTENTIAL EFFECTS OF MEDICARE ON MORTALITY

We use the model developed by Lleras-Muney and Moreau (2022) to structure our hypotheses about how Medicare's introduction may have affected mortality and longevity. Parental health and genes, as well as *in utero* conditions, endow individuals with an initial level of health $H \sim N(\mu_H, 1)$ that is normally distributed in the population with a standard deviation of one. Thereafter, health evolves at each age a as follows:

$$H_a = H_{a-1} - \delta a^{\alpha} + I + \varepsilon_a$$

Health grows due to the constant per-period average health investments I; it deteriorates with age at an increasing rate (given by the aging function δa^{α} with $\alpha > 1$); and it receives normally distributed shocks $\varepsilon_a \sim N(0, \sigma)$. This additive law of motion for health means that ε_a captures both direct health shocks and random variation in investments. Individuals die when their health falls below a certain threshold, $\underline{H}$, which is normalized to zero. In addition, a share κ randomly dies of accidental deaths each period after age 16. Despite its simplicity, this model has been shown to provide an excellent description of the mortality of populations from birth to death in stationary environments. It also quantitatively delivers log mortality curves that are linear in age after a certain age, consistent with observations by Gompertz (1825).

The model's seven parameters $(\mu_H, \delta, \alpha, I, \sigma, \kappa, \underline{H})$, define just a handful of channels through which Medicare may change mortality. Medicare may affect health investments (I) through both a price effect and an income effect arising from its health insurance structure, or equivalently, it may shift the health shock distribution. An alternative view of improved access to preventive and diagnostic care is that Medicare may slow the rate of aging (δ or α). Medicare may also lower the variance of health or health investment shocks (σ) for several reasons. Its insurance function transfers resources from good states (when individuals pay for premiums) to bad states when they suffer costly health shocks that are now covered by insurance, resulting in lower health in good times but higher health after bad health shocks. Medicare also reduces the financial burden of health shocks, which may help to maintain health investments, which are also reflected in ε_a. Finally, by leading to more and better acute medical care, Medicare may lower the threshold for dying ($\underline{H}$) keeping alive individuals who would otherwise die. Medicare cannot affect initial health, μ_H, and we assume that it does not affect mortality from accidental causes, κ (Bhaumik et al. 2023).

Figure 2a plots the simulated effect on a cohort's Gompertz curve from changes to each of these parameters. A ten percent increase in health investments (panel A.i) or a 5 percent decrease in the depreciation rate (panel A.ii) at age 65 simply makes the Gompertz curves flatter;[8] log mortality rates do not shift immediately, but they do rotate down so that mortality rises more slowly as people age. The reason mortality effects do not show up immediately but become visible over time is that a lot of individuals in the population will receive a boost in their health (or slow down the rate of aging), but because they are far from the death threshold, these changes do not affect mortality rates. By contrast, a decline in the variance of health shocks from 1 to 0.6 (panel A.iii) or the threshold for dying from 0 to -1 (panel A.iv), leads to immediate downward shifts in log mortality rates and then steeper slopes thereafter because the less healthy surviving cohort is very sensitive to subsequent shocks. In the long run, however, log mortality rates are higher under a smaller shock variance because while negative shocks are less negative, positive shocks as also less positive.

The differences between the two Gompertz curves from **Figure 2a**, which we plot in **Figure 2b**, are the treatment effects resulting from a change in each parameter. These predictions motivate our empirical approach because they show that dynamic treatment effects *within a cohort*, specifically the intercept and the slope of log mortality after cohorts gain Medicare eligibility, are crucial for making inferences about whether and how the program affects life expectancy.

Research on Medicare and mortality, however, looks at short-run effects for narrow age groups. Contemporaneous studies using the age-discontinuity in Medicare eligibility necessarily identify causal effects *at* age 65, and then only find immediate mortality reductions for severely ill patients (Card, Dobkin, and Maestas 2009). Research on the extension of Medicare to people with end-stage renal disease (in 1972) and the addition of prescription drug coverage (in 2006) finds reductions in mortality, but only within 4 to 6 years after reforms (Andersen 2018; Dunn and Shapiro 2019; Huh and Reif 2017). Existing research on Medicare's introduction focuses on five- or ten-year age groups and comes to mixed conclusions (Chay, Kim, and Swaminathan 2010; Finkelstein and McKnight 2008). None of these studies estimates longer-run mortality effects within cohorts needed to quantify Medicare's effect on life expectancy, nor are they able to measure mortality for subgroups other than by race, sex, or geography.

[8] There are two aging parameters. The effects of changing either are similar so we only display one.

In fact, randomized trials of the kinds of care available at the time do suggest longer-run effects. For example, the MRFIT randomized intervention, which aimed to lower cholesterol, lower blood pressure, and reduce smoking, only generated mortality declines after ten years, though they remained insignificant (Multiple Risk Factor Intervention Trial Research Group 1982; Multiple Risk Factor Intervention Trial Research Group 1990; Multiple Risk Factor Intervention Trial Research Group 1996). Clinical trials have shown that cholesterol-lowering drugs reduce all-cause mortality over 20 years (Ford et al. 2016), but these effects are not detectable within the first five years (Ray et al. 2010). Smoking cessation trials show declines in mortality that are significant after 14 years but not detectable earlier (Anthonisen et al. 2005). Medicare also improves access to diagnostic and preventive services, which could also benefit individuals with a delay (for evidence on colonoscopies, see (Niikura et al. 2017). Health insurance has also been shown to improve health behaviors and mental health in the short run (Baicker et al. 2013), which could lower mortality eventually.

In sum, the model predicts that Medicare could have large long-term effects, some of which may not be detectable in the short run and which empirical approaches used thus far may have missed. Additional empirical evidence suggests that the benefits of medical care could increase over time at least when measured by mortality rate declines. We estimate these dynamic benefits using various approaches next.

IV. DATA SOURCES AND OUTCOME MEASURES

IV.A. Data Sources

Census-Tree data. Our primary individual-level data come from the 1940 full-count census linked to information available from FamilyTree at FamilySearch, referred to hereafter as the Census-Tree. The 1940 full-count records, released on April 2, 2012, are publicly available on the Integrated Public Use Microdata Series (IPUMS) website (Ruggles et al. 2024). FamilySearch resources "help millions of people around the world discover their heritage and connect with family members" (FamilySearch 2025). It has a website that provides access to historical record collections and a wiki-style platform, called Family Tree, through which individuals can gather information about their ancestors. In 2020, there were over 1.2 billion individual profiles on Family Tree and over 12 million registered users, making it one of the largest genealogy websites in the world (Price et al. 2021). FamilySearch profiles contain information about deceased individuals, including sex, race/ethnicity, date of birth, place of

birth, date of death, and state of death, along with sources attached to support the validity of this information. We link the census records with profiles on the Family Tree using a matching file that FamilySearch shares with us. The 1940 Census includes variables such as education, income, and place of residence.

Social Security Administration cohort tables. These tables track the mortality and life expectancy of men and women in the US for cohorts born in 1900 and thereafter (Social Security Administration 2020). These data are only available at the aggregate (national) level by sex. These national-level cohort tables are constructed using death counts by five-year age groups from published vital statistics data pertaining to death registration deaths only, matched to population estimates constructed by the Census Bureau based on Census data (for 1900-1967). Because the vital registration system of the US was not complete until 1933, the SSA data are not necessarily representative of the nation for these years. We use them to assess the quality of the Census-Tree data and to estimate the structural model, which requires data from birth to death.

IV.B Sample Construction and Sample Selection

We linked the 1940 full-count census, which includes 132 million people, to the Family Tree. We obtain a sample of the 49 million individuals that are linked – this process we followed to construct the sample is described in **Figure A.1**. We then restrict attention to individuals born between 1885-1915 – these cohorts are estimated to be extinct and so we should observe age at death for everyone. We only keep observations with valid birth and death dates.[9] We further restrict our main analysis to whites because their coverage in the Family Tree database is more complete than for other race groups (Price et al. 2021) and because Medicare affected access to medical facilities for Black Americans in a much different way than it did for white Americans (Anderson, Charles, and Rees 2024; Smith 2016).

The final data set includes 18,485,813 individuals, accounting for 32.2% of our target population: white 1940 Census respondents born between 1885 and 1915. Men in our sample

[9] We rely on the year of birth recorded in the Family Tree in our analysis because the 1940 Census does not ask birth year and only reports age ("Enter the age of the person at his last birthday before 12:01 a.m., April 1, 1940."). As a result, the imputed birth year may not equal the actual birth year. However, the birth year variable from Family Tree was scraped because people would have had the chance to find birth records and make corrections.

live to age 72.9 on average, and women live to age 79.1, although the 90% percentile of the year of death in the full sample is 1994 for men and 2000 for women.

IV.C Outcome measures

Our sample includes people, i, who belong to a birth cohort $C_i = c \in [1885,1915]$ and die at discrete age $Y_i = a \in [40,115]$. Ages, periods, and cohorts are related by the identity $a = t - c$, and in our data these deaths necessarily occur in time periods $t \in [1940, 2023]$. We observe 1940 covariates X_i, and stratify our analysis by sex so all quantities in this section implicitly apply to men and women as measured in 1940.

The age-specific mortality rate for cohort c equals the probability of dying at age a conditional on surviving to age a:

$$\tilde{q}_{c,a} \equiv \frac{P(Y_i = a|C_i = c)}{P(Y_i \geq a|C_i = c)} = \frac{F_c(a) - F_c(a-1)}{1 - F_c(a)} \ . \qquad (1)$$

$F_c(\cdot)$ is the c.d.f. of Y_i among members of cohort c. The dynamics of $\tilde{q}_{ca}$ thus come from the shape of $F_c(\cdot)$.[10] We work with log mortality rates $q_{c,a} \equiv \ln(\tilde{q}_{c,a})$, and denote measured log mortality rates in our sample by $\bar{q}_{c,a}$.

IV.D Representativeness and Quality of Census Tree Data

A crucial concern with the data we create represents the population we aim to study. **Table 1** shows that our sample (first column) appears to be representative of the target population (Whites) in the 1940 census (last column) in terms of year of birth, ethnicity, veteran status, occupation score and educational levels.[11] However, it includes poorer households, fewer women, more married people, fewer people either born or residing in the Northeast in 1940.

We correct the observable imbalance by re-weighting the linked sample to make it representative of the target population (Bailey et al. 2020; Lleras-Muney, Price, and Yue 2022; Yue et al. 2023). Specifically, we estimate the probability, $p(X_i)$, of a successful match

[10] This one-year mortality rate, $\tilde{q}_{c,a}$, equals the integral of the continuous-time hazard between age a and $a + 1$. Exact dates of birth and death are not always accurate, so we measure mortality rates in discrete units and use life-table methods to calculate life expectancies.

[11] When estimating the probability of matching we use the birth year variable from the Census so we can observe cohort for matched and unmatched people and use it to construct the weights. We then restricted the Census-Tree sample to those whose birth year falls within two years of their Census birth year, which it drops 5.3% of the Census-Tree sample (see **Figure A.1**). Therefore, the Census-Tree sample of 1885-1915 cohorts matches to birth cohorts 1883-1917 in IPUMS 1940 census.

between our sample and the 1940 full-count Census as a function of the covariates using a *robit* model (Newson and Falcaro 2023; Seaman and White 2013).[12] When constructing mortality rates from the Census-Tree data, we reweight matched observations by $p(X_i)^{-1}$. The second column of **Table 1** shows that, as expected, reweighting brings the sample covariate means substantially closer to the target population means (**Tables A.1a and A.1b**).

To assess the quality of death information in our data, we compare the weighted age-specific mortality rates in our data with those in the SSA cohort life tables and those in the Human Mortality Database (HMD) cohort death rates for the U.S. HMD data includes death rates starting in 1993 for all our studied birth cohorts, 1885-1915 (Human Mortality Database 2023). **Figure A.2** shows that the Census-Tree mortality rates are comparable to the SSA life tables and HMD cohort death rates for all birth cohorts and for both men and women, albeit slightly lower, particularly in old age. This likely occurs because our Census-Tree sample excludes Black people and underrepresents immigrants, both of whom had higher mortality rates than white Americans. As a result, **Figure 3** shows that life expectancy at age 65 is 0.5 to 1 year higher in the Census-Tree sample than in the SSA and HMD data. Notably, the trends in life expectancy at age 65 across birth cohorts are similar between the Census Tree and HMD data for both men and women. For men, life expectancy remains relatively flat for the 1885-1900 cohorts and then gradually increases for the 1900-1915 cohorts. For women, it first steadily increases between the 1885 and 1905 cohorts and then plateaus for the subsequent 1906-1915 cohorts.

V. EMPIRICAL STRATEGY

V.A Potential outcomes and target parameters

The objects of interest in this paper are causal parameters that describe Medicare's effect on mortality rates or functions of those parameters that describe its effect on life expectancy. To define them, we write potential outcomes in terms of the time and the age at which one first gains Medicare. Units from cohort c become treated by Medicare at time $G_t(c) = max(c + 65, 1965) = g_t$ which occurs when they are age $G_a(c) = max(65, 1966 - c) = g_a$. The only people who gained Medicare after age 65 necessarily got it in 1966, and the only

[12] In addition, we further truncated the weights at the 99th percentile (Seaman and White 2013). We include the following covariates (X_i) available in the 1940 full-count census: sex, race, ethnicity, marital status, years of schooling, household total income, family size, labor force participation, occupation income scores, year of birth, state of residence, and place of birth. We estimated the model separately by sex.

people who gained Medicare after 1966 necessarily got it at age 65. The values g_t and g_a define potential outcomes for age-at-death (Robins 1986; Rubin 1974): $Y_i(g_t, g_a)$. We use $Y_i(\infty)$ to denote potential outcomes in the absence of Medicare. Potential mortality rates $\tilde{q}_{c,a}(g_t, g_a)$ have the same form as (1) but use potential instead of realized outcomes.

Our building block target parameters are average treatment effects on the log mortality rates of each cohort at each age:[13]

$$ATT(g_t, g_a, a) \equiv q_{c,a}(g_t, g_a) - q_{c,a}(\infty) \qquad (2)$$

Since Medicare "treats" all members of a cohort we take the phrase "on the treated" to refer to all members of a cohort exposed to Medicare in year g_t at age g_a. This parameter reflects many different channels through which Medicare may have affected mortality: the direct increase in (meaningful) insurance coverage and health care use, the program's effect on health system capacity or inputs (Finkelstein 2007), technology (Clemens and Olsen 2021), or prices (Clemens and Gottlieb 2014). The collection of $ATT(g_t, g_a, a)$s are *within-cohort* event-study effects that fully characterize the effect of Medicare's existence on mortality and lifespan. Because there are too many to report individually, we summarize them in three intuitive ways.

First, we report average treatment effects by event-age, $e \equiv a - g_a$, to show within-cohort dynamic effects as in **Figure 2b**:

$$ATT(e) \equiv \sum_{g_a=65}^{A} \sum_{g_t=1966}^{T} w(g_t, g_a) \times ATT(g_t, g_a, g_a + e) \ . \qquad (3)$$

The weights $w(g_t, g_a)$ equal the share of the population who survived to their Medicare age who are in the (g_t, g_a) cohort. This answers the question: how did Medicare affect mortality rates of its beneficiaries on average in the years after they gained eligibility?

We report cross-cohort heterogeneity by summarizing each cohort's post-Medicare $ATT(g_t, g_a, a)$s with an intercept term ($\delta(g_t, g_a)$) and a slope term ($\theta(g_t, g_a)$). These are simply weighted averages that equal coefficients from a regression of each cohort's $ATT(g_t, g_a, a)$ on a constant and e for $e > 0$:

$$\delta(g_t, g_a) \equiv \sum_{e=0}^{A} \frac{ATT(g_t, g_a, g_a+e)}{A+1} - \theta(g_t, g_a)\left(\frac{g_a+A}{2}\right) \qquad (3a)$$

[13] We use log mortality rates for several reasons. First, parameters defined in logs are easier to interpret across ages with different mortality rates (Deaner and Ku 2024). Second, for reasons discussed below, we believe that identification of log parameters is credible, while other transformations are not (Roth and Sant'Anna 2023).

$$\theta(g_t, g_a) \equiv \frac{\sum_{e=0}^{A} ATT(g_t, g_a, g_a+e)\left(e-\frac{A}{2}\right)}{\sum_{e=0}^{A}\left(e-\frac{A}{2}\right)^2} \qquad (3b)$$

Cohorts born before 1901 gained Medicare eligibility after age 65, and are the only cohorts in Medicare's history to gain access later in life. Therefore, heterogeneity in $\delta(g_t, g_a)$ and $\theta(g_t, g_a)$ across g_a is relevant to policy questions about changes to Medicare's eligibility structure. The model in section III also suggests that their magnitudes provide information about how Medicare affects health production.

Finally, we use life-table methods to combine observed and counterfactual mortality rates ($q_{c,a}(\infty) \equiv q_{c,a} - ATT(g_t, g_a, a)$) to construct average effects of Medicare on life expectancy at age 65:

$$ATT_{LE}(g_t, g_a) = E[Y_i(g_t, g_a) - Y_i(\infty) | G_t(C_i) = g_t, G_a(C_i) = g_a, Y_i \geq 65] \qquad (4)$$

It is important to compute life expectancy gains in addition to impacts on mortality because the former is shaped both by Medicare's causal effects on mortality and the level of mortality (Dahl et al. 2024). Thus a group that experiences the largest drop in mortality is not necessarily the group that will experience the largest gains in life expectancy.

Both empirical evidence and the model of health capital accumulation discussed in section III point to potential cross-sectional heterogeneity in the effects of health insurance on mortality. Our linked data uniquely allow us to target causal effects by education and income subgroups. These are comparable to the parameters just defined, but involve mortality rates for subgroups in 1940, $\tilde{q}_{c,a}(x) \equiv \frac{P(Y_i = a | C_i = c, X_i = x)}{P(Y_i \geq a | C_i = c, X_i = x)}$.

V.B Identification and estimation

We rely on three distinct assumptions on untreated log mortality rates to identify the $ATT(g_t, g_a, a)$ parameters: the full structure of the model from section III, linearity of untreated log mortality rates, and a parallel trends assumption across cohorts. All three strategies require an assumption that Medicare does not affect mortality before a cohort gains eligibility.

Assumption NA. No anticipation

For unit i from cohort $C_i = c$ eligible for Medicare at age g_a in year g_t:

$$Y_i(g_t, g_a) = Y_i(\infty) \quad if \quad Y_i(g_t, g_a) < g_a$$

Assumption NA says that anyone who dies before reaching Medicare age in a world with Medicare, would have died at the same age without Medicare. This implies that $q_{c,a}(g_t, g_a) = q_{c,a}(\infty)$ and thus $ATT(g_t, g_a, a) = 0$ for $a < g_a$, which makes pre-Medicare mortality informative about counterfactual mortality rates. Violations of this assumption could arise if people anticipate their own eligibility, if Medicare has general equilibrium effects that alter mortality of not-yet-eligible people, or if some people get Medicare before age 65.[14] Below we discuss how anticipation may affect our estimates and we test for it.

V.B.1 Structural Design

Our first approach uses the model in section III to estimate counterfactual mortality rates. For each cohort, we estimate all of the model's parameters (normalizing $\underline{H} = 0$) using data from birth until the age they qualify for Medicare. We make only one modification to the baseline model: we allow for *I* to be different in 1918 to account for the excess mortality associated with WWI and the flu pandemic; $\hat{I}_{flu}$. We then simulate mortality through age 95 using these values and construct counterfactual log mortality rates, $\hat{q}_{c,a}^{SIM}(\infty)$ as a function of the estimates $(\hat{\mu}_H, \hat{\delta}, \hat{\alpha}, \hat{I}, \hat{I}_{flu}, \hat{\sigma}, \hat{\kappa}, 0)$. We estimate the model using the Simulated Method of Moments, and target the differences in the survival curve as the objective function. (see **Appendix Section 1** for more details).

The red line in **Figure 4** plots log mortality rates predicted by the model for the 1902 men cohort with parameters estimated on data through age 65. The blue line plots observed log mortality rates. The gap between the lines after age 65 is the treatment effect estimator for this cohort:

$$\widehat{ATT}^{SIM}(g_t, g_a, a) \equiv \bar{q}_{c,a} - \hat{q}_{c,a}^{SIM}(\infty)\,. \quad (5)$$

The validity of this approach depends on the correct specification of the model and the ability to estimate its parameters using pre-Medicare data. The model embeds several strong assumptions, such as constant parameters and no optimization.[15] Nevertheless, it successfully fits patterns of mortality for a wide range of populations and replicates the observed effects of

[14] Today, people on the Social Security Disability Insurance program, or who have end-stage renal disease, or who are widows or widowers of a Medicare-eligible spouse can get Medicare before they turn 65. None of these provisions were in place until 1973, when the 1908 cohort aged into Medicare.

[15] Optimizing models with these features are well understood, but without detailed data on health, incomes, and investments they are not estimable (e.g. Khwaja 2010).

high SES or of temporary or permanent shocks to health occurring at some point in the lifetime (Lleras-Muney and Moreau 2022).

We also face several limitations estimating the model's parameters. The most practical challenge is that the model requires data on complete life tables from birth to age 65, which means we can only use the SSA life tables for cohorts born in 1900 and after. Moreover, shocks earlier in life, such as the 1918 flu, can affect mortality trajectories later in life, biasing our model-based counterfactuals. We account for the flu in our estimation, but as Lleras-Muney and Moreau (2022) discuss, these shocks are generally difficult to model. The objective function is also poorly behaved, so the estimates are sensitive to initial guesses. To address this, we first estimate the model for the 1900 birth cohort repeatedly until we can no longer improve the fit. Then we use these parameter values as initial guesses for subsequent cohorts.

We assess the model's fit by estimating a linear trend through the difference between observed and predicted log mortality at ages 55-64 (see **Tables A2a and A2b**, which also report mean squared error). If the model estimates are on average unbiased then the predicted and observed mortality will be very similar up to age 65 and the trend term will be statistically insignificant. We find that the model fits well for men born between 1900 and 1909, but poorly for younger cohorts of men and for all cohorts of women. We therefore only report structural results for men born before 1909 and we do not present estimates for women.

V.B.2 Interrupted Time-Series Design

Our second approach is an interrupted time-series design based on a functional form assumption on untreated log mortality rates, $q_{c,a}(\infty)$.

Assumption GM (Gompertz-Makeham). Linearity of log untreated mortality rates

For all cohorts, c, and ages between a_{c0} and a_{max}, the log of untreated period mortality rates is linear in age:

$$q_{c,a}(\infty) = \alpha_c + \beta_c(a - a_{c0}) \qquad (6)$$

This standard Gompertz-Makeham model is also implied by the theoretical model and supported by extensive empirical evidence (e.g., Chetty et al. 2016) since Gompertz first observed it as an empirical regularity in 1825. This regularity however holds only among adults of a certain age as can be seen for the 1902 birth cohort in **Figure 4**.

Under assumptions NA and GM, α_c and β_c can be obtained by regressing $q_{c,a}$ on a constant and age for each cohort using pre-Medicare ages $a \in [a_{c0}, g_a]$. Denoting the coefficients from

this regression by $\tilde{\alpha}_c$ and $\tilde{\beta}_c$ leads to the following estimand for counterfactual mortality rates at post-Medicare ages: $\tilde{\alpha}_c + \tilde{\beta}_c(a - a_{c0})$. The dashed lines in **Figure 5** plot these linear estimates of counterfactual mortality rates and the solid lines plot observed log mortality rates.

This motivates an interrupted time-series (ITS) plug-in estimator that uses our sample mortality rates $\bar{q}_{c,a}$to estimate $\tilde{\alpha}_c$ and $\tilde{\beta}_c$ (denoted $\hat{\alpha}_c$ and $\hat{\beta}_c$) and then constructs $ATT(g_t, g_a, a)$:

$$\widehat{ATT}^{ITS}(g_t, g_a, a) \equiv \bar{q}_{c,a} - \hat{\alpha}_c - \hat{\beta}_c(a - a_{c0}) \qquad (7)$$

These are labeled as the difference between the solid grey line (observed) and dashed blue line (counterfactual) in **Figure 5**. This estimator yields $ATT(g_t, g_a, a)$ parameters until age a_{max}, the age at which mortality is no longer linear.

Unlike the model-based estimates that use the full history of mortality to identify the structural parameters, the ITS approach relies only on the assumption GM during ages close to Medicare eligibility. Key to this approach then is to determine the range of ages, a_{c0} to a_{max}, during which log-linearity is appropriate. We set $a_{max} = 90$, similar to Chetty et al. (2016) and Fletcher et al. (2022), which avoids bias from non-linearities in log mortality and mismeasurement of age at death for the oldest old.

The choice of a_{c0} is less clear and likely differs by cohort and sex (as can be seen in **Figures A.3a and A.3b**). We use our 20% training sample to fit a series of two-slope models to log mortality rates before Medicare eligibility, selecting a_{c0} as the best fitting trend-break using five-fold cross-validation (Card, Mas, and Rothstein 2008).[16] We do this separately for six cohort groups (1885-1889, 1890-1894, 1895-1899, 1900-1905, 1906-1909, and 1910-1915). **Figure A.4** plots the selected a_{c0} over birth cohorts (see **Appendix Section 2** for more details).

V.B.3 Difference-in-Differences Design

Our third approach is a difference-in-differences (DiD) design based on an assumption about trends $q_{c,a}(\infty)$.[17]

[16] To gauge the fit of a linear model for estimated Gompertz curves, we regress the log of mortality on age for each birth cohort by sex and report the corresponding R^2 for pre-trends, which we define as trends in log mortality from the age in 1940 to the age before Medicare eligibility age. Specifically, for birth cohort $c \in (1885,1915)$, the age window for pre-trend is: $max(40, 1940 - c)$, $max(65,1966 - c) - 1$. **Figure A.5** shows that a linear model fits the Gompertz curves almost perfectly for males with an R^2 over 0.98 for all included birth cohorts. The model fit for females is also excellent for old cohorts 1885-1887 (R^2=0.99) but gradually decreases across cohorts to 0.945 for the 1904 cohort, then increases to over 0.98 for later cohorts 1910-1915.

[17] Assumption PT is also a proportional hazards assumption because it is equivalent to a constant the ratio of age-specific mortality rates between cohort c and the average comparison cohort (Deaner and Ku 2024).

Assumption PT. Parallel Trends across cohorts and ages

The change in untreated log mortality rates for cohort c between ages $G_a(c) = g_a$ and a equals the change in log untreated mortality rates between the same ages for older cohorts, ℓ, treated after age a and between 1 and $(k-1)$ years after age $G_a(c)$; that is, for all ℓ such that $max\{a, g_a\} < G_a(\ell) < g_a + k$:

$$q_{c,a}(\infty) - q_{c,g_a-1}(\infty) = q_{\ell,a}(\infty) - q_{\ell,g_a-1}(\infty)\,. \qquad (8)$$

Assumption PT involves a cohort treated at age g_a and comparison cohorts treated "later." In our case, later means "at a later age", so the comparison cohorts are older.[18] A larger value of k means a larger yet older comparison group that identifies parameters at older ages (**Figure A.6**).[19] Assumption PT says that these cohorts' Gompertz curves would have been parallel through age a in the absence of Medicare.

Figure 6 illustrates how the PT assumption identifies counterfactual mortality changes in a simple case where we compare two cohorts at a time. To identify $ATT(1967,65,65)$, which is the effect of Medicare on mortality for the 1902 cohort in the year they entered Medicare (i.e., at age 65 in 1967), we compare their change in mortality between ages 64 and 65 to the change in mortality for older cohorts at the same ages. In **Figure 6**, we show this for a comparison cohort born in 1900 (ages 64 and 65 in 1964 and 1965) or in 1891 (ages 64 and 65 in 1955 and 1956). Identification of $ATT(1967,65,74)$, the effect for the 1902 cohort at age 74, works the same way but requires a comparison cohort born at least ten years earlier (in this case, 1891).

Under assumptions NA and PT, a simple DiD estimand identifies $ATT(g_t, g_a, a)$. Plugging in sample analogues yields the following DiD estimator (where ℓ is defined in Assumption PT and the weights w_ℓ reflect the relative size of the comparison cohorts measured at age $g_a - 1$):

$$\widehat{ATT}^{DiD}(g_t, g_a, a) = \left(\bar{q}_{c,a} - \bar{q}_{c,g_a-1}\right) - \sum_{\ell} w_\ell \left(\bar{q}_{\ell,a} - \bar{q}_{\ell,g_a-1}\right) \qquad (9)$$

This is a version of the Callaway and Sant'Anna (2021)'s estimator, which we implement by adding the choice of k to the Stata command `csdid` (Rios-Avila, Sant'Anna, and Callaway

[18] The max (a, g_a) notation ensures that all $(k-1)$ comparison cohorts contribute to the pre-treatment falsification tests when $a < g_a$.

[19] Assumption PT defines comparison cohorts as those whose Medicare age comes after $G_a(c)$, which means that for cohorts treated later and by definition at age 65, the only available comparison cohorts are those born in up to $(k-1)$ years before 1901. Therefore, the cohort gap between treatment and comparison units grows as we look at younger cohorts in this design.

2023), defining cohorts as the cross-sectional unit, age as the time variable, and $G_a(C_i)$ as the treatment variable.

Intuitively assumption PT is more likely to be satisfied for cohorts that are closer together in age than for cohorts that are not. For example, WWI and the 1918 flu had different mortality effects across cohorts may lead to non-parallel slopes in log mortality thereafter (Lleras-Muney and Moreau 2022). To select k, we use our 20% training sample and estimate event-study parameters $ATT_k(e)$ using comparison cohorts defined by values of k between 7 and 15. We first measure the estimated pre-trends and find that the linear pre-trend does not vary strongly with k; it is always insignificantly positive (**Figure A.7** panel A). We then consider robustness to parallel trends violations using the smoothness restriction method of Rambachan and Roth (2023). For each k, we find the maximum bias that still allows us to reject H_0: $ATT_k(5) = 0$. Here we find clearer guidance: $k = 12$ allows us to reject this null under the largest worst-case bias (**Figure A.7** panel B). See **Appendix Section 3** for further details.

V.B.4 Estimating aggregated parameters

We report aggregates of the estimated $\widehat{ATT}(g_t, g_a, a)$ parameters as described in section V.A. Both the event-study ($\widehat{ATT}(e)$) and the cohort-specific intercept ($\hat{\delta}(g_t, g_a)$) and slope ($\hat{\theta}(g_t, g_a)$) aggregations are linear combinations of the building block parameters and thus simple to calculate using post-estimation commands.

We use life table methods described in **Appendix Section 4**, to estimate $\widehat{ATT}_{LE}(g_t, g_a)$ parameters. Because the structural model identifies counterfactuals at all ages, we have estimates $\widehat{ATT}^{SIM}(g_t, g_a, a)$ for each cohort's entire post-Medicare lifespan. An advantage of the model is that it captures commonly observed non-linearities in later life mortality; these are particularly important for accurately estimating effects on life expectancy.[20] When calculating life expectancy effects based on the ITS and DiD designs, which we only estimate within an age window around Medicare, we assume that the estimated linear trend across ages in each cohort's $\widehat{ATT}(g_t, g_a, a)$ parameters continue until age 90 and that counterfactual mortality evolves in parallel to observed mortality thereafter (**Figures A.8 and A.9**). We use cohort specific changes in intercept and slope of log mortality to compute the implied effect on life

[20] In particular, there is a debate about whether log mortality rates continue to be linear in very old age or instead decelerate (e.g., Feehan 2018). In this model mortality rates decelerate in old age due to selection (individuals who have had many positive shocks survive to the oldest ages). Some argue that this deceleration is due to poor and sparse data quality among the oldest old (e.g., Gavrilov and Gavrilova 2019) but there is much debate about whether this is the case (e.g., Alvarez et al. 2021).

expectancy at age 65 separately for each cohort. We assume everyone dies by age 115 to close the life table. To obtain the 95% confidence intervals of ITS and DID implied effects on life expectancy at age 65, we bootstrap the estimates with 1000 resamples.

VI. THE EFFECT OF MEDICARE ON MORTALITY AND LIFE EXPECTANCY

VI.A Average results

Figure 7 presents event-study estimates of Medicare's causal effects on mortality. For men, we find clear evidence that Medicare reduced log mortality rates (panel A). The pre-period falsification tests are only significant in one case, never larger than 0.05, and display no obvious trend, even up to 10 years prior to Medicare in the model-based estimates. After gaining Medicare eligibility, however, the results from all three approaches are negative, clearly distinguishable from zero after two years, and growing in magnitude linearly over time. After ten years, we find that Medicare reduced age-specific mortality rates for white men by about 14 percent ($\widehat{ATT}(9) \approx -0.15$), an estimate that is remarkably similar across designs. The structural estimates show that these declines level off after ten years, but remain large ($\widehat{ATT}^{SM}(20) \approx -0.14$) 20 years after cohorts gain Medicare. This result highlights the value of the model since it would be hard to predict this pattern based on the reduced form results alone.

A simple trend break, at least over ten years, summarizes these event-study results well. The slope change is -0.013 (s.e. = 0.001) for ITS and -0.019 (s.e. = 0.003) for DiD (**Table A.3**). The ITS estimates also have a small intercept shift (-0.021, s.e. = 0.006), but the DiD estimates do not (0.011, s.e. = 0.011). Although the structural model can only be estimated for a subset of cohorts (1900-1909 cohorts), the results are similar (-0.017 change in the slope and -0.012 change in the intercept). Through the lens of the model, this pattern suggests that, for men, Medicare is equivalent to increasing health investments or lowering depreciation/aging rates.

These ITS and DiD estimates translate into an average gain in life expectancy for men of 0.87-1.20 years (**Table 2**). Our confidence intervals rule out changes smaller than 0.72 years, a meaningfully large increase in longevity. The structural model suggests gains of about 0.97 years for the 1900-1909 cohorts. The (unweighted) average of the effects for the same cohorts is about 1.5 years using both ITS and DiD. The difference likely come from the fact that we compute the ITS and DiD life expectancy estimates by assuming that the effects on mortality

would continue to grow linearly until age 90, whereas the model suggests that the effects plateau after 10 years.

The aggregate event-study estimates for women shown in **Figure 7b** are not as conclusive as the men's results. The ITS estimates are essentially zero in the first five years and turn *positive* and significant thereafter. The DiD estimates are also insignificant in the first six years and then become *negative*, but at the ten year mark they are less than half as large as the results for men. The ITS and DiD point estimates suggest that pre-Medicare mortality rates were roughly linear and comparable across cohorts, but they are imprecise (the standard errors of these estimates are much larger than those for men) and cannot rule out potentially meaningful bias (in either direction). Recall that we cannot present results using the structural model because the model does a poor job at fitting mortality among the elderly in the decade prior to age 65.

The inconclusive effects for women are also apparent in their estimated intercept and slope changes (**Table A.3).** The ITS estimator yields statistically significant declines in the intercept and increases in the slope, whereas the DiD estimator does not indicate a change in the intercept but does show a statistically significant decline in the slope. The two methods, therefore, give wildly different estimates for women's life expectancy: ranging from a significant increase of 0.87 years using the DiD estimates to a significant *decline* of 0.80 years using ITS (**Table 2**).

VI.B Results by birth cohort

Figure 8 plots intercept and slope estimates ($\hat{\theta}_c$ and $\hat{\delta}_c$) for each cohort by sex. For men, the cohort-specific results are generally quite close to the aggregate results. For cohorts born before 1908 or so, both reduced-form methods estimate a precise slope change of about 2 percent per year, while most intercept estimates are statistically insignificant. The estimates that are available from the structural model are typically indistinguishable from those we obtain from the ITS approach. This similarity across cohorts is striking because the older cohorts are the only cohorts in US history who first received Medicare at ages other than 65, some as late as age 81. The results in **Figure 8** show that Medicare affected the path of log mortality similarly for these groups.

We observe a significant change in the results for the most recent birth cohorts of men. First, the estimated intercepts become negative and statistically significant in the ITS design for cohorts born 1908 and later. We also see that the ITS slopes become insignificant for cohorts born after 1910 and turn positive for the 1914 and 1915 cohorts. The cohort-by-cohort checks

of the identification strategy suggest this is occurring because our identification assumptions fail for these later cohorts (see **Figures A.11-A.15, and Appendix Section 5**). First, the structural model cannot match the pre-Medicare mortality rates for these cohorts because there are non-linearities occurring before age 65 for these younger cohorts, with mortality rates increasing more slowly than linearity would predict. When we estimate the ITS and DiD models by birth cohort groups we indeed find that there are significant pre-trends for younger cohorts, but not for other subsets of cohorts (**Figure A.14**). These patterns are especially pronounced for the 1914 and 1915 birth cohorts, whose coverage in our Family Tree sample (which ended in 2023) was not yet complete due to the practice of requiring additional death verification for anyone who would not yet be 110 years old. Our final results will use complete data for these cohorts.[21] Excluding the 1909-1915 birth cohorts, our average effects on life expectancy are similar to those presented in **Table 2** at around one year of life using both methods (**Table A.4)**. These reduced form results for the cohort born after 1908 or so align with the failure of the structural model to match mortality in old ages prior to age 65 and suggest that there are other factors affecting mortality prior to Medicare that the model cannot match because we have not accounted for them, and which also result in a failure of the reduced form identification approaches.

Figure 9 shows the estimated effects on LE at age 65 ($\widehat{ATT}_{LE}(g_t, g_a)$) by cohort. The results are very consistent across designs for men. Medicare leads to small gains in LE for men born in 1885, but its effects grow for younger cohorts. The estimates peak at around 2 years of life for the 1906 cohort (ITS) and the 1906-1908 birth cohorts (DiD). They also peak at 1.30 years of life for the 1906-1907 cohorts in the structural model. Because the effects on mortality rates are similar across most cohorts in all designs, as **Figure 8** shows, this phase-in pattern comes from the differing length of time that cohorts spent on Medicare. Cohorts that became eligible earlier in life experienced lower mortality rates for longer, which generated greater gains in lifespan. The average effect on life expectancy for cohorts obtaining Medicare in 1966 after

[21] There are two major policy changes that could also explain these results. The first is the change in 1972 to Medicare which made individuals under the age of 65 with long term disabilities or with end stage renal disease eligible for the program starting in 1973. The second is the Clean Air Act of 1970, which started operating in 1971 and lead to significant reductions in air pollution. Because these changes occurred after 1966 and might have affected individuals prior to age 65, these two changes might explain why our identification assumptions fail for the most recent cohorts of men since they could result in a decline in the age profile of mortality even before age 65 leading us to underestimate the impact of Medicare. They could also lower mortality rates after age 65 which might inflate the estimates of the effects of Medicare.

age 65 (for cohorts born before 1901) is around 0.68. If we focus on the 1901-1909 birth cohorts, which got Medicare at age 65 (similar to today) and for which the identification assumptions appear to be satisfied, then we obtain an average gain of 1.55 in the reduced form approaches and about 1 in the structural model. This highlights that conclusions based on mortality rate changes cannot necessarily be applied to life expectancy.

For women, **Figures 8** and **9** show that both the ITS and the DiD approaches suggest null effects on the life expectancy of women from older cohorts but conflicting and mostly statistically insignificant effects on life expectancy for younger birth cohorts. We discuss these inconclusive results for women in section VII.

VI.C The effects of Medicare by education and income

Figure 10 plots the event-study estimates by sex and for the three education groups separately: those with less than elementary school (0-7 years of schooling), those who graduated from elementary school but not high school (8-11 years of schooling), and high school graduates (12+ years of schooling). These correspond to 24, 47 and 29% of men in the population, and 21, 46 and 33% of women in the population (**Table 2**). For men, the ITS and DiD event-study estimates by education are almost identical to the aggregate results, especially in the short run. By year 9, however, the ATT estimate is about -0.2 for men (or larger) with 0-7 years of schooling, but about -0.12 for men with 12 or more years of schooling. Thus over the longer run the mortality impacts appear larger for less educated men. For women, we find mixed results. Women with 0-7 years of school have the largest mortality reductions using ITS but while negative the effets are statistically insignificant. Using DiD all groups appear to have declines but the effects are not larger for women with less education.

Figure 11 presents event-study estimates by income terciles based on household income in 1940. The event studies show relatively little heterogeneity by income for both methods and both sexes, though unexpectedly the group in the middle income group appears to have the largest effects except for men in the DiD design. This may come from the fact that income in our data is measured 26 years before Medicare begins and may therefore be a poor proxy for lifetime income (see Haider and Solon 2006) or income at the time we estimate treatment effects. Our coarse cut of the income distribution and use of cohort-specific ranks, however, should mitigate this measurement error.

These heterogeneity estimates are interesting for at least two reasons. First, it is not obvious whether Medicare should have larger or smaller health effects on higher-income or more-educated people. Higher-SES groups might gain less from Medicare because they were more likely to have insurance before 1966 (Epstein and Murray 1967) and were in better health (Braveman et al. 2010; Kitagawa and Hauser 1973). On the other hand, conditional on having insurance high-SES people visit specialists at higher rates (Braveman et al. 2010; Dunlop, Coyte, and McIsaac 2000) and may be more likely to take advantage of medical advances (Glied and Lleras-Muney 2008).[22] We do not find strong heterogeneity along these margins.

Second, the subgroup estimates relax our identification assumptions. Differential mortality rates by education can generate compositional changes that vary with age, making assumptions GM or PT fail even if they hold within education or income groups.[23] The stratified analyses in **Figures 10** and **11** nonparametrically control for these trends, and the results do not change.

A subtle issue arises when translating these mortality effects into life expectancy gains: groups who see the largest decreases in mortality rates may not see the largest increases in life expectancy if they have elevated mortality rates (Dahl et al. 2024).[24] **Table 2** documents estimated effects on life expectancy that decrease with education levels, but by less than the mortality effects do. We find that Medicare increased life expectancy on average by about 1-1.2 years for men with less than a primary education, but by between 0.67 (ITS) and 0.85 (DiD) years for high school graduates. These differences are not statistically significant from each other, so on net the effects on life expectancy at age 65 do not appear to be meaningfully higher for the less educated despite their larger declines in mortality rates.

[22] The model reflects this ambiguity as well. Ex-ante it is not clear that groups with initially high levels of investment or low levels of depreciation would benefit more or less in terms of life expectancy. More people in high-initial-health groups reach 65, but the survivors from a low-initial-health group are very positively selected, making it unclear which surviving group is healthier and would benefit more from Medicare.

[23] Educational levels in our data increased substantially—the percent of men (women) with 12+ years of schooling increased from 19.1% (20.6%) for the 1885 birth cohort to 44.6% (49.2%) for the 1915 cohort, consistent with prior studies (Goldin 1998; Lleras-Muney 2005). Education is also associated with lower mortality rates in these cohorts (Goldin 1998; Lleras-Muney 2005; Lleras-Muney, Price, and Yue 2022; Yue et al. 2023).

[24] To gain intuition about this result consider the following example. Suppose that mortality rates λ are constant at every age and the time until death follows an exponential distribution so that life expectancy is given by $1/\lambda$. Suppose that in period 1 group A has a mortality rate $\lambda_a = 0.1$ and group B has a mortality rate of $\lambda_b = 0.2$, so that life expectancy is equal to 10 for group A and 5 and group B. Suppose now that mortality falls, more so for group B which has the initially higher rates. In the second period $\lambda_a = 0.06$ and $\lambda_b = 0.1$, so the decline in mortality is larger for B in absolute and relative terms (the decline for B is of 0.1 or 50%, whereas for A the decline is only of 0.04 or 40%). But the life expectancy of A is now 16.6 and that of B is 10. Group A sees an increase of 6.6 years whereas B only sees an increase of 5 years despite their larger mortality declines. This occurs because the mortality rates for B are higher than A's in period 2.

For women, the results are again different across methods. The ITS only estimates positive gains for the lowest education group but these are statistically insignificant. The effects for the other two groups are negative. So using ITS the effects are more positive for the lowest education group but none of the effects are positive. The DiD on the other hand estimates larger effects for the most educated group, but the effects are positive and not statistically significantly different from each other.

Table 2 also shows that men from low-income and high-income households have very similar estimated life expectancy effects, with the largest estimates for middle income men but the differences across income groups are not statistically significant. This pattern is similar for women in the DiD design but in the ITS design all the estimates are negative. Moreover we cannot reject the null that all the estimates are identical.

VI.D Spillover estimates at younger ages

After just 18 months, Medicare accounted for over 10 percent of personal health expenditures in the US (CMS, 2025). Medicare's introduction was also associated with increases in hospital entry, capacity, technology adoption (Finkelstein 2007), and medical device patenting (Clemens and Olsen 2021). To the extent that these changes also affected mortality rates, our $ATT(g_t, g_a, a)$ estimates include spillovers. A key *interpretation* question, then, is to what extent the estimates reported above reflect spillovers versus the direct effect of Medicare coverage? In addition, the $ATT(g_t, g_a, a)$ estimates for cohorts that gained Medicare after 1966 (1902-1915) would also be biased by spillovers because they use at least some post-1966 data to estimate counterfactuals. Spillovers, therefore, also affect the *validity* of our designs.

While we cannot separately identify spillovers and direct effects using our treated cohorts, our historical context allows us to estimate spillover effects on younger cohorts before they turn 65 and age into Medicare themselves. **Appendix Section 6** motivates ITS and DiD designs that identify spillover effects by looking for mortality changes in 1966 younger for cohorts who gained Medicare *later* ($G_t(c) > 1966$).

Figure 12 plots event-study estimates from both approaches, neither of which finds any evidence that Medicare's spillovers affected mortality for the near elderly. The point estimates are small and never statistically significant for up to 7 years after Medicare. Even in our 20% sample, we can rule out an average spillover effect on log mortality before age 65 of more than -0.035 using DiD.

These findings are consistent with the evidence in **Figure 1** that neither insurance coverage nor hospital use increased substantially right after Medicare's introduction among younger people. Of course, Medicare's most important spillovers may have taken time to materialize or may have been larger for the over-65 population if insurance itself was necessary to access hospital capital investments or new technologies. Nevertheless, Medicare's documented spillovers on the health system do tend to appear within just a few years (Finkelstein 2007). Moreover, **Figure 1** shows that around 80 percent of people ages 55-64 had private insurance in 1968, suggesting that the population for whom we estimate spillovers had ways to pay for these new services. Therefore, early effects of Medicare on the health care system appear not to have had important mortality effects on younger people. This supports an interpretation of our main estimates as direct effects of Medicare's coverage, though we cannot rule out spillovers that occurred only *within* the elderly population.

VI.E Assessing the validity of the identification assumptions

We find clear evidence that Medicare's introduction reduced men's mortality rates gradually over at least ten years, inconclusive evidence about its effects on women, and little evidence that these operated through spillover effects, at least when measured among slightly younger people. Given the structure of our estimators, however, changes to (untreated) mortality rates around 1966 are an important threat to internal validity. In fact, our spillover estimates already suggest that there are not large calendar time effects in untreated mortality rates. The results in **Figure 12** do not just reflect spillovers, they also capture differential changes in untreated mortality rates that arise in 1966 (see the assumptions in **Appendix Section 6.ii**). The null findings in **Figure 12** fail to produce evidence of important time shocks.[25]

A different set of concerns is that the passage of Medicare coincided with economic, policy, or environmental changes that affected the over 65 population differently than the under-65 population. We now discuss the largest and most likely such shocks.

*Anticipation***.** All estimation approaches rely on the assumption that mortality does not respond prior to becoming eligible. This seems reasonable for the cohorts that were first treated in 1966 because the passage of Medicare was difficult and unanticipated, as discussed in Section II. Cohorts born in 1902 and after, though, clearly knew about Medicare and might, for example,

[25] It is possible that the results in **Figure 12** come from offsetting spillover effects and time shocks, although we view this as unlikely.

delay non-urgent care until age 65.[26] We generate evidence on this using a *t*-test of the equality of the average DiD estimate in the two years prior to reaching Medicare age ($\widehat{ATT}^{DiD}(g_t, g_a, g_a - 2)$) for the 1902-1915 cohorts versus the 1885-1901 cohorts. We do not detect a difference in pre-Medicare mortality effects for cohorts that could anticipate coverage (The difference for men is 0.03, 95% CI: -0.01, 0.07).[27] We also tried omitting the two years before Medicare eligibility from the calculation of the Gompertz slope in our ITS design and obtained nearly identical results (**Figure A.17**). Thus we can rule out short term anticipatory effects.[28]

Medicaid. Medicaid was created at the same time as Medicare, and covered the same basic health services as well as nursing home care and, in most states, drugs (De Lew 1995). Medicaid receipt also increased at age 65 because welfare benefits, which were the statutory basis of Medicaid eligibility, began at that age for older people. By 1976, about 14% of people 65 and older received Medicaid (US Census Bureau 1992). For almost all of them, Medicare was the first payer, so Medicaid provided *incremental* benefits, including paying Medicare's deductibles, cost sharing, and Part B premiums, and covering nursing homes and drugs.

Several pieces of evidence, however, suggest that Medicaid cannot seriously confound our mortality estimates. Most importantly, Medicaid was far too small in the over-65 population, especially relative to Medicare, for its incremental coverage to account for our findings. **Appendix Section 7** shows that for Medicaid to explain our effects entirely, its marginal services and financial protection would have to reduce mortality by at least 41%. This is 10 times as large as recent estimates of the causal effect of losing subsidized drug coverage through Medicaid among elderly people who retain Medicare (Roberts et al. 2025). Using a 4 percent effect size, our event-study estimate at time 5 would change from -0.084 to -0.76; not a statistically significant difference.[29]

---

[26] Card, Dobkin and Maestas (2008) find that elective procedures rise at age 65 particularly for previously uninsured individuals consistent with patients delaying care. Similarly, Decker (2005) finds that mammography use increases sharply at age 65. Clemens and Gottlieb (2014) document that physicians' financial incentives within the Medicare program also strongly influence the provision of elective services.

[27] We computed the difference as $ATT_{e=-2,g_a==65} - ATT_{e=-2,g_t=1966}$ and bootstrapped it with 1000 resamples to obtain the 95% bias corrected confidence interval.

[28] We cannot rule out however that the most recent cohorts changed their savings and work behavior many years before becoming eligible.

[29] It is also 4 times as large as the effect on the treated of Medicare part D from Huh and Reif (2017). We conducted similar calculations assuming that Medicaid's causal effects came from the 2.5% of people over 65 who use Medicaid-funded long-term care. If Medicaid *caused* all of them to use the nursing home, and their untreated mortality rates were ten times as high as the non-institutionalized population (approximately 35% per year, relative

Further, our estimates themselves do not suggest an important role for Medicaid. The similarity of our effects on mortality across the income distribution contrasts with Medicaid's income-based targeting. In 1976, for example, 21% of seniors in the bottom third of the income distribution received Medicaid, compared to 10% of seniors in the top two-thirds (U.S. Census Bureau. 2006). We also estimate nearly identical ATT parameters for groups of states that introduced Medicaid in different years: 1966, 1967-1969, or 1970. **Figure A.18** plots event-study estimates using ITS and DiD for each state group. Even though no seniors in the 1970 states received Medicaid for the first 5 event times, we find that Medicare's mortality effect is the same as it is in the states where the two programs corresponded in time.

Income trends. Real median incomes rose by more than half during the 1950s, a period when much of our study population was still working. Congress also raised Social Security benefits several times during the late 1960s and early 1970s, which affected pension income for all our cohorts. **Appendix Figure A.19** uses Census and CPS data to quantify within-cohort income trends, which may also influence the mortality patterns we leverage in our design. Real median family income grew across cohorts at essentially every age, but every cohort experienced a substantial reduction in income in their mid-60s, when many workers retired.

Could our results be attributed to these trends instead of Medicare? The literature on causal effects of income on mortality suggests not. The most closely related research comes from a reduction in Social Security benefits for the 1917 cohort. Snyder and Evans (2006) find that this reform *lowered* mortality by about two percent.[30] The key mechanism for this effect appears to be that lower benefits increased labor supply which may have kept "seniors connected to the community and reduce[d] social isolation" (pg 493). Subsequent research on early Social Security concludes the same (Fitzpatrick and Moore 2018). Applying these results to observed trends in labor force nonparticipation within cohorts, which we plot in **Appendix**

to about 3.5% for the over-65 population at this time), then Medicaid's nursing home care would have had to reduce their mortality by 36% (-0.45 log points) to explain our main findings. Neither these conditions nor this effect size is plausible. Many Medicaid nursing home residents were previously institutionalized in state hospitals or private nursing homes, so Medicaid's causal effect on nursing home use must be smaller than 2.5 percent. Second, one study found that in 1976 the mortality rate of nursing home residents was at most 2.5 times the rate in the rest of the population. Finally, recent evidence using a random "judge" assignment design in the Netherlands finds no average effect of nursing home admission on mortality (Bakx et al. 2020).

[30] Handwerker (2011), however, argues that this contrast reflects cross-cohort mortality trends, while Noghanibehambari and Fletcher (2025) conclude that lower Social Security benefits raise mortality at later ages. Schwandt (2018) documents that plausibly random stock market fluctuations lead to relatively higher survival rates for respondents in the Health and Retirement Study who hold more stock.

Figure A.20, implies that income and retirement trends should generate a fairly large *increase* in mortality later in life—the opposite of our results.

Our data also fail to support retirement as a source of bias. Most directly, we find no evidence of changes in the slope of mortality at age 65 among older cohorts who received Medicare later in life. Using the 1885-1896 cohorts ($g_a \in [70,81]$), the estimated trend-break in log mortality for men is just 0.0029 (s.e.=0.0057); small even relative to the pre-65 Gompertz slope of 0.078. If retirement strongly reduced mortality (the *opposite* direction of much of the related literature) we would expect two patterns in our results. Estimates for older cohorts who gained Medicare later should be smaller than estimates for younger cohorts for whom Medicare often coincides with retirement (Rust and Phelan 1997). The DiD estimates, which difference out mortality changes for older cohorts around retirement, should also be substantially smaller than the ITS and structural estimates which estimate counterfactual mortality using only pre-retirement information. In fact, we fail to find support for either of these predictions.[31]

VI.F Combining short term reduced form estimates with structural estimates

The previous discussion rules out many specific alternative interpretations for our findings, but the longer the evaluation period, the more likely it is that other changes in the environment begin to matter. For example, our estimates could reflect general medical progress in the 1960-2000 period. Previous research concludes that about one-third of the declines in cardiovascular mortality during the second half of the 20th century were due to innovations such as the dissemination of knowledge about the harms of smoking, the diffusion of hypertension and later cholesterol drugs, and the development of invasive treatments such as bypass surgery and angioplasty (Cutler, Landrum, and Stewart 2009; Cutler and Meara 2003).[32] Our estimates will be biased if these changes affect our treated cohorts but not the older data we use to construct counterfactuals (though it may well be that health insurance and new technologies complemented each other during this period since health insurance increased access to these

[31] The prosperity of the 1950s could generate bias if rising income *levels* affect later-life changes in Gompertz slopes. Our model shows that this can occur if rising income protects cohorts from health-related deaths (which cause Gompertz curves to slope up) until increasingly older ages. Again, this is the opposite sign of our main results and is thus not a plausible alternative explanation. Simulations also show that while higher health investments shift the Gompertz curve once health-related deaths begin to bite, this does not alter its slope. Vital Statistics data also fail to show big changes in external cause deaths across these cohorts. In 1950, for example, the external-cause share of deaths for 50–54-year-olds (1896-1900 cohorts) was 11.5% and in 1958 (1905-1908 cohorts) it was 10.9%, even though median family income at these ages grew by about 30% across these cohorts.

[32] It is unclear whether we would want to "net out" these effects, though, because these innovations might not have diffused in the absence of Medicare (Clemens and Olsen 2021; Finkelstein 2007).

novel but expensive technologies, in which case our estimates are not biased but would differ in environments with lower medical innovation).

To overcome this issue, we follow the approach of Deryugina and Reif (2023) to calculate longer-run effects based only on short-run causal estimates. First, we use the 5 or 10 year short-term reduced form estimates to estimate the change in the model parameters that rationalizes the declines in mortality implied by these estimates. We assume that these short-term estimates are not contaminated by other aggregate shocks. If this is the case, then the changes in the model parameters are unbiased and can be used to predict the effect of the program. So, in a second step, we use these estimated parameter changes to predict the effect of Medicare on life expectancy. We do this by computing what life expectancy would be if the parameters remained at their pre-Medicare level, and alternatively, if Medicare changed the parameters at age 65 in exactly the way that is needed to match the reduced form evidence. We match the reduced form estimates using the three parameters that can result in the type of declines that the reduced form produces, namely I, α, or δ. We only conduct this exercise for men for whom we have consistent reduced form results and reliable structural model estimates.

Table 3 shows that all parameters can fit the reduced form end point similarly well, though δ or α appear to generate the best fit for both time horizons, suggesting that declines in the rate of aging best characterize the effects of Medicare. The implied changes in life expectancy from changing each parameter are similar: they hover around 0.6 years of life if we match the 5-year end point, and about 1.7 year of life if we match the 10-year end point.

Additionally, note that the 5-year estimates are smaller than the 10-year estimates. If we want to be conservative and avoid attributing to Medicare the effects of other determinants of elderly mortality that arose after the early 1970s, then the 5-year estimates are the most trustworthy. Nevertheless, even these conservative estimates are significant. Life expectancy at age 65 rose by about 1.5 years for men across the cohorts we study. These computations suggest that Medicare can explain at least one-third of the gains across all 31 cohorts.

VII. DISCUSSION

Our findings suggest that, on average, Medicare reduced men's mortality rates by 15 percent after ten years and their life expectancy at age 65 by one year. The results for women, however, are inconclusive. This section discusses the plausibility and interpretation of the magnitude and pattern of these estimates.

VII.A Discussing sex differences

Our results for men are consistent across empirical approaches, but do not generally even have the same sign for women. Several commonly cited reasons for different results by sex cannot account for this. Our Family Tree data contain maiden names, so we measure women's mortality well (see **Table 1** and **Figure 3**). Insurance rates and hospital use also respond similarly to Medicare for men and for women (see **Figure 1**), so the differences do not arise from differential effects on medical care use. What could explain these patterns?

Our data do show one clear difference between the men and women in our sample: cohort life expectancy for women over 65 was already rising prior to the introduction of Medicare. This is shown in **Figure 3** and contrasts sharply with the case of men. Black et al. (2023) show similar patterns for life expectancy at age 25. Thus some factor prior to age 65 clearly benefited the cohorts of women we study. Unless these patterns were entirely due to health endowments, this fact could explain why our structural model fails: it cannot fit the profile of mortality well unless the environment is stable, or the environmental changes, such as the 1918 flu, are explicitly accounted for. Unfortunately, it is unclear why the life expectancy of women rose for these cohorts (e.g., Goldin and Lleras-Muney 2019), and it is thus unclear how to account for these changes in estimation.[33] Note, however, that these changes would tend to bias the SM and ITS approaches more than DiD to the extent that the cross-cohort comparisons successfully capture these hard-to-model trends. This is consistent with the closer concordance between men's and women's results using DiD.

Alternatively, women's mortality trends could be linked to the experiences of their male family members, who are almost always older and less healthy husbands. For example, if wives' mortality starts to fall when their husbands gain Medicare eligibility, our model fit would suffer, and the pre-Medicare slope used to form the ITS counterfactual would be too flat. To investigate this, we re-estimate the model for women who were never married.[34] Our results are similar for this group (**Figure A.21**), ruling out this as an explanation for our results. Women are also much more likely than men to experience the death of a spouse between the

[33] One possibility is that the spread of sulfa drugs and the enormous reduction in the health risks associated with childbirth changed adult mortality profiles for women specifically. The rise in women's life expectancy, however, also occurs for cohorts that were already too old to benefit from these innovations.

[34] Our Family Tree data allows us to retrieve all the spouses of a woman. I define those without a spouse recorded in the Family Tree as "never married." We also find a similar null effect for those reported as "single or not married" from the 1940 census.

ages of 45 and 75 (our critical window): 53% of women in our data become widows, compared to only 20% of men. Widowhood is associated with a significant temporary increase in mortality, which doctors often refer to as "broken heart syndrome" (Ennis and Majid 2021; Moon et al. 2011). We observe this in our data (**Figure A.22)**. We do find that accounting for this widowhood effect significantly lowers the positive ITS estimates among married women for whom we observe the age at death of their spouses (**Figure A.23**). If the distribution of the age at which women become widows is stable across cohorts, then the DiD estimates are less susceptible to this bias, as shown in **Figure A.24**.

Based on this discussion, we might want to conclude that only the DiD estimates for women are reliable. They are less susceptible to biases from unmodeled shocks or non-linearities that are common across cohorts, they are robust to corrections for some of these shocks, and they qualitatively match the results for men. These estimates are still substantially smaller than the results for men. In fact, this is consistent with research showing that many new medical technologies available during this period were not particularly effective for women. As Thomas and Braus (1998) noted, *"Until a decade ago, men were the model subjects in most funded biomedical studies (…). It was then assumed that whatever the findings, the results would hold true in women. Since then, it has become apparent that this generalization was incorrect in many situations."* The Hypertension Detection and Follow-Up study, a 10,500-person clinical trial of anti-hypertensive drugs conducted in the 1970s, found reductions in five-year mortality of between 15 and 28 percent for men and Black women, but no effect on white women (Hypertension Detection Follow-up Program Cooperative Group 1979).

In sum, we cannot fully explain why our results for women are consistent with the men's results using DiD but not ITS. The health and lifespan of women appear to have been improving earlier in the century for reasons that are not fully understood and are therefore difficult to address. But the historical evidence also suggests that the smaller benefits of Medicare may have been because innovation in cardiovascular disease was much less beneficial for women than men. We thus cannot draw strong conclusions about how Medicare affected women.

VII.B Magnitudes on the treated population

An important piece of evidence on the validity of reduced-form effects of health insurance reforms on mortality involves the size of the population whose "treatment status", typically defined by insurance coverage or health care consumption, was causally affected by the policy.

Since Medicare cannot more than eliminate mortality, comparing our estimates to the size of a "complier" population provides a natural plausibility check (see Goodman-Bacon 2018).[35]

Yet measuring the size of such a complier population is hard for two reasons. First, Medicare shifted people from uninsured to insured, from privately insured to publicly insured, and even led some people to hold *both* public and private policies.[36] We cannot estimate separate treatment effects for each type of complier (Kline and Walters 2016), and so must make additional assumptions to justify evaluating the plausibility of our effect sizes by appealing to a single complier population. Second, Medicare may have induced complex changes to treatment (i.e., insurance) *paths*, and neither our demographic nor potential outcomes model takes a stand on how one's insurance history affects current health (e.g., Goldin, Lurie, and McCubbin 2021). Thus, measuring the size of the complier population and the intensity of treatment over longer time horizons depends on further assumptions about the model for outcomes.

Two important features of our context suggest a relatively large "insurance complier" population. First, no one lost Medicare; once seniors were exposed to Medicare, they were always treated with public hospital insurance at least. Second, given the structure of insurance markets at the time, it is reasonable to assume that once someone lost insurance, they were never again treated with private insurance. This is consistent with the steady decline in the pre-Medicare age profile of insurance in 1963 (**Figure 1**).

Assuming that without Medicare, insurance rates for the elderly would have changed in parallel with those for younger people, then we can use **Figure 1** to construct several "first stage" measures. We first adjust the post-Medicare insurance rates among seniors to account for underreporting. Gindi and Cohen (2012) report that Medicare reporting among respondents 65 and older in the 2005 NHIS has a false positive rate of 2.8% and a false negative rate of 89%, which implies a relatively constant Medicare participation rate in our data of about 96%. Next,

[35] This is an extreme criterion. Many effect sizes that are smaller than a 100% reduction in mortality are often considered unreasonably large. In fact, our model suggests that even Medicare eliminated health-related morality (which is itself an implausible claim), accidents would still cause deaths.

[36] Here we discuss insurance as "the" treatment, but one could also view health care use or the use of specific effective health care services as the treatment that Medicare shifted. In fact, evidence from randomized trials suggests our estimates are plausible. Heart disease was the main cause of death among elderly men above in 1965. Reductions in hypertension and cholesterol and more aggressive interventions for heart attack patients have been estimated to increase life expectancy for men by several years (Cutler, Landrum, and Stewart 2009; Wright and Weinstein 1998). Medicare could easily have increased the use of these kinds of clinically effective services.

we first compute the change in insurance rates between 1963 and 1968 at each age and subtract the average change for people 55-64. This yields estimated insurance changes between 22 (age 65) and 64 (age 84) percentage points.

If compliance means that Medicare *ever* caused one to be insured instead of uninsured, the size of the complier population at age 75 is just the effect on age 75 insurance: 39.8 percentage points. Using this to scale our estimate $\widehat{ATT}(9)$ yields a causal effect on compliers of -35% (-0.14/0.398). This would be smaller for older cohorts, however, who gained more insurance but had similar treatment effects. On the other hand, if the underlying treatment concept is the change in the number of years of insurance, then it is appropriate to sum the insurance effects across ages, which yields a gain of 3.3 years by age 75 (but more than twice that, 8.4 years, by age 85). This suggests an effect per year of insurance of -4.5 percent. Both of these effect sizes are in line with estimates from Medicaid's introduction, which reduced mortality among treated children by 31 percent (Goodman-Bacon 2018) and cumulative mortality into adulthood by about between 6 and 8 percent per year of childhood coverage (Goodman-Bacon 2021).

VII.C Cost-benefit calculations

The fact that we estimate benefits from Medicare does not necessarily imply that the program was worth it, because, as noted earlier, it is very expensive. To better assess this, we now estimate the cost of the program per life saved and compare it to estimates of the statistical value of a year of life. The gains we estimate are large enough to justify expenditures per enrollee at the time. The estimated annual cost per enrollee in 1970 was around $368 (about $3,000 in today's dollars) (Buntin et al. 2003). If men enrolled in Medicare at age 65 lived for 15 years after age 65 (14 years + 1 gained), the total cost per enrollee's lifetime and of the additioan life-year gained is about $45,000. This is much lower than a central estimate of the statistical value of a year of life ($150,000; Keller et al. 2021). Today's cost per beneficiary is substantially larger however, at about $15,000, making the cost of an extension of a year of life close to $225,000.

However, the cost of these life extensions is larger than the cost of similar life extensions resulting from programs that benefit younger populations, such as cash transfers to poor families, youth training programs, or Medicaid.[37] On the other hand, the comparable cost from

[37]Aizer et al. (2016) find that giving about 30-50% of FPL in cash to poor families for a median duration of 3 years increased the LE of boys by 1.5 years. The cost of that was up to 50% of the FPL per year. The FPL today for a family of 5 is $37,650, so the total cost was $56,475 per family, or ~14,120 per kid (4 kids and a mom,

directly providing health care through Community Health Centers was $54,000 (Bailey and Goodman-Bacon 2015).

VII.E Comparison with other studies of Medicare

Our approach and results contrast in several ways with two closely related studies that compare changes in age-specific mortality across calendar time for people over 65 versus under 65. Finkelstein and McKnight (2008; FM) find little evidence that log mortality for people aged 65-74 fell after 1966 relative to mortality for people aged 55-64.[38] Chay, Kim and Swaminathan (2010; CKS), on the other hand, report clear evidence that mortality rates (and log mortality rates) for people aged 65-69 fell relative to those for people aged 60-64.

There are two key distinctions between our analysis and the ones in FM and CKS. The first lies in how we treat cohorts. In a time series of age-specific mortality rates, time and cohort effects are collinear, so shifts in mortality profiles due to pre-Medicare factors like early life conditions or the changes in lifetime income (see **Appendix Figure A.19**) are a potential source of bias. In contrast, our design only uses *within-cohort* mortality changes to identify Medicare's effects, which eliminates cohort effects. (Of course, in our set-up, age and time are collinear, but, as discussed above, **Figure 12** does not suggest that this biases our findings.)

Our analysis also has an important measurement advantage: we observe exact denominators for our mortality rates. Both FM and CKS use highly accurate Vital Statistics data on deaths, but must construct population denominators. FM apply a cubic interpolation to decennial population counts by state and age from 1950-1980, while CKS combine intercensal (1950s) and provisional (1960s and after) estimates for the whole country. **Appendix Figures A.25** and

assuming all money spent on kids). Thus, the cost of 1 year life extension was $9,412. Aizer et al. (2024) find that training men ages 19-25 for about 1 year during the great depression in the CCC program increased lifespan for about 1 year. The cost of the program estimated to be about $23,000. Thus, the estimated cost of a 1-year life extension was $23,000. Goodman-Bacon (2021) finds that the cost to gain one per quality-adjusted life year through childhood Medicaid coverage in the 1960s was about $9,000.

[38] FM also use a special tabulation of 1963 insurance rates by 11 sub-regions in the NHIS. They compare changes in age-specific mortality rates before and after 1966 in sub-regions that had different health insurance coverage rates in 1963 and find that mortality actually *rose* slightly in areas where Medicare led to larger coverage gains. There are two major limitations to interpreting this contrast as a causal effect of Medicare. First, DiD designs with a continuous treatment variable (and no untreated units) require a kind of treatment effect homogeneity to identify an interpretable causal parameter (Callaway, Goodman-Bacon, and Sant'Anna 2024). If regional health insurance coverage was correlated with factors that potentially mediate the effect of Medicare on mortality, this assumption need not hold. Consistent with this, CKS show that elderly hospital utilization grew by the same amount in the North (a low insurance gain region) and the South (a high insurance gain region) after 1966, suggesting that mortality effects should be comparable as well. Second, the 1963 NHIS sample design was only intended to be representative at the region level (and separately by urban residence). Random measurement error in sub-regional uninsurance rates will attenuate estimates from a continuous DiD design.

A.26 replicate both findings using Vital Statistics data as well as our data. Within about 5 years of Medicare's passage, our data as well as the results in FM and CKS suggest a reduction in young-elderly mortality of about 3 percent. Those figures also show, however, that Vital Statistics based estimates are extremely sensitive to the measurement of population denominators. Using linearly interpolated denominators, for instance, eliminates the null longer-run results that led FM to conclude that Medicare was ineffective. Using intercensal estimates almost entirely eliminates the relative trend break in log mortality documented in CKS. Since the choice and measurement of denominators is not obvious, a key strength of our approach is that this choice is not necessary.

VIII. CONCLUSIONS

This paper uses a large and novel dataset that measures lifetime mortality for a wide range of cohorts and includes individual economic and demographic characteristics measured in 1940. We use three pre-specified research designs—one based on a structural model (Lleras-Muney and Moreau 2022), an ITS design, and a DiD design—to estimate dynamic within-cohort causal effects of the introduction of Medicare on log-mortality rates. For white men, all three approaches show clear evidence that Medicare reduced mortality rates across essentially all cohorts born between 1885 and 1905, and that they generated cohort-level changes in life expectancy of up to 1.5 years.

We do not find the same to be true for women, for whom the estimates are more unstable. Whether this is the result of identification failures or not is unclear. However, it is also true that medical understanding of how to treat cardiovascular disease for women was inadequate in the 1960s perhaps explaining why we fail to find effects. These large sex differences necessitate further investigation, but they are not unique to our study.

What are the implications of our study for today? It is difficult to apply our estimates to current populations because the technology available today and coverage for people under 65 is different from what was available when our study population lived and died. However, we can use our results to simulate gains from Medicare for today's cohort under different scenarios. Raising the age of Medicare's eligibility has been considered as a means to lower Medicare expenditures. We estimate that an increase in the age of eligibility to age 75 would result in a loss of 0.3 to 1 years of life, relative to gaining Medicare at age 65 (**Table 3**). Because Medicare is popular, others have proposed lowering the age of eligibility instead, making the program less targeted to the old. We estimate that lowering eligibility to age 55 would instead lead to

life expectancy gains at age 55 of about 0.4 to 1.4 year higher than provision at age 65. Future work could use our estimates to conduct alternative counterfactuals to assess whether these changes would be desirable.

Future work should also investigate effects on health. There are a number of valuable health interventions that improve the quality of life without extending life, particularly among older populations (e.g., Kavalieratos et al. 2016). These include palliative care, chronic pain management, hearing and vision aids hip replacement, and perhaps mental health interventions. Our estimates do not quantify these important benefits. With estimates of the health impacts, our estimates of life expectancy benefits, and existing estimates of the financial benefits of Medicare, a comprehensive assessment of Medicare would be possible.

OPPORTUNITY AND INCLUSIVE GROWTH INSTITUTE, FEDERAL RESERVE BANK OF MINNEAPOLIS

UNIVERSITY OF CALIFORNIA, LOS ANGELES, AND NATIONAL BUREAU OF ECONOMIC RESEARCH

BRIGHAM YOUNG UNIVERSITY AND NATIONAL BUREAU OF ECONOMIC RESEARCH

UNIVERSITY OF MARYLAND, COLLEGE PARK

REFERENCES

Aizer, A., S. Eli, J. Ferrie, and A. Lleras-Muney, "The Long-Run Impact of Cash Transfers to Poor Families," *American Economic Review*, 106 (2016), 935-971.

Aizer, Anna, Nancy Early, Shari Eli, Guido Imbens, Keyoung Lee, Adriana Lleras-Muney, and Alexander Strand, "The Lifetime Impacts of the New Deal's Youth Employment Program," *Quarterly Journal of Economics*, 139 (2024), 2579-2635.

Aizer, Anna, Hilary Hoynes, and Adriana Lleras-Muney, "Children and the US social safety net: Balancing disincentives for adults and benefits for children," *Journal of Economic Perspectives*, 36 (2022), 149-174.

Alvarez, Jesús-Adrián, Francisco Villavicencio, Cosmo Strozza, and Carlo Giovanni Camarda, "Regularities in human mortality after age 105," *PloS one*, 16 (2021), e0253940.

Andersen, M. S., "Effects of Medicare coverage for the chronically ill on health insurance, utilization, and mortality: Evidence from coverage expansions affecting people with end-stage renal disease," *Journal of Health Economics*, 60 (2018), 75-89.

Anderson, D Mark, Kerwin Kofi Charles, and Daniel I Rees, "Imposing policy on reluctant actors: The hospital desegregation campaign and Black postneonatal mortality in the Deep South," *Review of Economics and Statistics*, (2024), 1-46.

Anthonisen, Nicholas R, Melissa A Skeans, Robert A Wise, Jure Manfreda, Richard E Kanner, John E Connett, and Lung Health Study Research Group*, "The effects of a smoking cessation intervention on 14.5-year mortality: a randomized clinical trial," *Annals of Internal Medicine*, 142 (2005), 233-239.

Baicker, K., S. L. Taubman, H. L. Allen, M. Bernstein, J. H. Gruber, J. P. Newhouse, E. C. Schneider, B. J. Wright, A. M. Zaslavsky, A. N. Finkelstein, and Group Oregon Health Study, "The Oregon experiment--effects of Medicaid on clinical outcomes," *New England Journal of Medicine*, 368 (2013), 1713-1722.

Bailey, M., C. Cole, M. Henderson, and C. Massey, "How Well Do Automated Linking Methods Perform? Lessons from U.S. Historical Data," *Journal of Economic Literature*, 58 (2020), 997-1044.

Bailey, Martha J, and Andrew Goodman-Bacon, "The War on Poverty's experiment in public medicine: Community health centers and the mortality of older Americans," *American Economic Review*, 105 (2015), 1067-1104.

Bakx, Pieter, Bram Wouterse, Eddy Van Doorslaer, and Albert Wong, "Better off at home? Effects of nursing home eligibility on costs, hospitalizations and survival," *Journal of Health Economics*, 73 (2020), 102354.

Barcellos, Silvia Helena, and Mireille Jacobson, "The effects of Medicare on medical expenditure risk and financial strain," *American Economic Journal: Economic Policy*, 7 (2015), 41-70.

Bhaumik, Deepon, Chima D Ndumele, John W Scott, and Jacob Wallace, "Association between Medicare eligibility at age 65 years and in-hospital treatment patterns and health outcomes for patients with trauma: regression discontinuity approach," *British Medical Journal*, 382 (2023).

Black, Bernard, Alex Hollingsworth, Leticia Nunes, and Kosali Simon, "Simulated power analyses for observational studies: An application to the affordable care act medicaid expansion," *Journal of Public Economics*, 213 (2022), 104713.

Black, Sandra E, Neil Duzett, Adriana Lleras-Muney, Nolan G Pope, and Joseph Price, "Intergenerational Transmission of Lifespan in the US," Working paper No. 31034. (Cambridge, MA: *National Bureau of Economic Research*, 2023).

Boudreaux, Michel H, Ezra Golberstein, and Donna D McAlpine, "The long-term impacts of Medicaid exposure in early childhood: Evidence from the program's origin," *Journal of Health Economics*, 45 (2016), 161-175.

Braveman, P. A., C. Cubbin, S. Egerter, D. R. Williams, and E. Pamuk, "Socioeconomic disparities in health in the United States: what the patterns tell us," *American Journal of Public Health*, 100 Suppl 1 (2010), S186-196.

Brown, David W, Amanda E Kowalski, and Ithai Z Lurie, "Long-term impacts of childhood Medicaid expansions on outcomes in adulthood," *Review of Economic Studies*, 87 (2020), 792-821.

Buntin, M. B., J. Escarce, D. Goldman, and et al., "Determinants of Increases in Medicare Expenditures for Physicians' Services," Working paper No. (Rockville (MD): 2003).

Callaway, Brantly, Andrew Goodman-Bacon, and Pedro HC Sant'Anna, "Difference-in-differences with a continuous treatment," Working paper No. 32117 (Cambridge, MA: *National Bureau of Economic Research*, 2024).

Callaway, Brantly, and Pedro HC Sant'Anna, "Difference-in-differences with multiple time periods," *Journal of Econometrics*, 225 (2021), 200-230.

Card, D., C. Dobkin, and N. Maestas, "The Impact of Nearly Universal Insurance Coverage on Health Care Utilization: Evidence from Medicare," *American Economic Review*, 98 (2008), 2242-2258.

Card, David, Carlos Dobkin, and Nicole Maestas, "The Impact of Nearly Universal Insurance Coverage on Health Care Utilization and Health: Evidence from Medicare," Working paper No. WR-197. (Santa Monica, CA: 2004).
---, "Does Medicare save lives?," *Quarterly Journal of Economics*, 124 (2009), 597-636.
Card, David, Alexandre Mas, and Jesse Rothstein, "Tipping and the Dynamics of Segregation," *Quarterly Journal of Economics*, 123 (2008), 177-218.
Caswell, Kyle J, and John H Goddeeris, "Does Medicare reduce medical debt?," *American Journal of Health Economics*, 6 (2020), 72-103.
Center for Health Administration Studies, and National Opinion Research Center, "Survey of Health Services Utilization and Expenditures, 1963," (Inter-university Consortium for Political and Social Research (ICPSR) [distributor], 1984).
National Health Expenditure Data (https://www.cms.gov/data-research/statistics-trends-and-reports/national-health-expenditure-data,
Chay, Kenneth Y, Daeho Kim, and Shailender Swaminathan, "Medicare, hospital utilization and mortality: Evidence from the program's origins," Citeseer, 2010. https://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.309.4678&rep=rep1&type=pdf
Chetty, R., M. Stepner, S. Abraham, S. Lin, B. Scuderi, N. Turner, A. Bergeron, and D. Cutler, "The Association Between Income and Life Expectancy in the United States, 2001-2014," *Journal of the American Medical Association*, 315 (2016), 1750-1766.
Chung, Andrea Park, Martin Gaynor, and Seth Richards-Shubik, "Subsidies and structure: the lasting impact of the Hill-Burton program on the hospital industry," *Review of Economics and Statistics*, 99 (2017), 926-943.
Clay, Karen, Joshua Lewis, Edson Severnini, and Xiao Wang, "The value of health insurance during a crisis: Effects of medicaid implementation on pandemic influenza mortality," *Review of Economics and Statistics*, 106 (2024), 1393-1402.
Clayton, Denise Hammock, "The Effect of Prescription Drug Coverage on Mortality: Evidence from Medicaid Implementation," *Journal of Health Economics*, 63 (2019), 100-113.
Clemens, Jeffrey, and Joshua D Gottlieb, "Do physicians' financial incentives affect medical treatment and patient health?," *American Economic Review*, 104 (2014), 1320-1349.
Clemens, Jeffrey, and Morten Olsen, "Medicare and the Rise of American Medical Patenting: The Economics of User-Driven Innovation," Working paper No. 9008. (2021).
Cunningham, Robert M., III, and Robert M. Cunningham, Jr., *The Blues: A History of the Blue Cross and Blue Shield System* (Dekalb, IL: Northern Illinois University Press, 1997).
Cutler, David, Mary Beth Landrum, and Kate Stewart, "Intensive Medical Care and Cardiovascular Disease Disability Reductions," in *Health at Older Ages: The Causes and Consequences of Declining Disability Among the Elderly*, (Chicago: University of Chicago Press, 2009).
Cutler, David, and Ellen Meara, "Changes in the Age Distribution of Mortality over the 20th Century," in *Perspectives on the Economics of Aging*, (University of Chicago Press, 2003).
Dahl, Gordon B., Claus Thustrup Kreiner, Torben Heien Nielsen, and Benjamin Ly Serena, "Understanding the Rise in Life Expectancy Inequality," *Review of Economics and Statistics*, 106 (2024), 566-575.
Dalgaard, Carl-Johan, and Holger Strulik, "Optimal aging and death: understanding the Preston curve," *Journal of the European Economic Association*, 12 (2014), 672-701.
De Lew, Nancy, "The first 30 years of Medicare and Medicaid," *Journal of the American Medical Association*, 274 (1995), 262-267.
Deaner, Ben, and Hyejin Ku, "Causal Duration Analysis with Diff-in-Diff," 2024. https://arxiv.org/abs/2405.05220
Decker, Sandra L, "Medicare and the health of women with breast cancer," *Journal of Human Resources*, 40 (2005), 948-968.
Deryugina, Tatyana, and Julian Reif, "The long-run effect of air pollution on survival," Working paper No. 31858. (Cambridge, MA: *National Bureau of Economic Research*, 2023).
Dunlop, S., P. C. Coyte, and W. McIsaac, "Socio-economic status and the utilisation of physicians' services: results from the Canadian National Population Health Survey," *Social Science & Medicine*, 51 (2000), 123-133.
Dunn, Abe, and Adam Hale Shapiro, "Does medicare part D save lives?," *American Journal of Health Economics*, 5 (2019), 126-164.
Ennis, Jeffrey, and Umair Majid, ""Death from a broken heart": A systematic review of the relationship between spousal bereavement and physical and physiological health outcomes," *Death Studies*, 45 (2021), 538-551.
Epstein, Lenore, and Janet Murray, *The Aged Population of the United States: the 1963 Social Security Survey of the Aged* (Washington, D.C.: Government Printing Office, 1967).

FamilySearch (https://www.familysearch.org/en/home/about,
Feehan, Dennis M, "Separating the signal from the noise: evidence for deceleration in old-age death rates," *Demography*, 55 (2018), 2025-2044.
Finkelstein, A., "The aggregate effects of health insurance: Evidence from the introduction of Medicare," *Quarterly Journal of Economics*, 122 (2007), 1-37.
Finkelstein, Amy, and Robin McKnight, "What did Medicare do? The initial impact of Medicare on mortality and out of pocket medical spending," *Journal of Public Economics*, 92 (2008), 1644-1668.
Fitzpatrick, Maria D, and Timothy J Moore, "The mortality effects of retirement: Evidence from social security eligibility at age 62," *Journal of Public Economics*, 157 (2018), 121-137.
Fletcher, Jason M, Michal Engelman, Norman J Johnson, Jahn Hakes, and Alberto Palloni, "Understanding geographic disparities in mortality," *Demography*, 60 (2022), 351-377.
Ford, Ian, Heather Murray, Colin McCowan, and Chris J Packard, "Long-term safety and efficacy of lowering low-density lipoprotein cholesterol with statin therapy: 20-year follow-up of West of Scotland Coronary Prevention Study," *Circulation*, 133 (2016), 1073-1080.
Gavrilov, Leonid A, and Natalia S Gavrilova, "New trend in old-age mortality: Gompertzialization of mortality trajectory," *Gerontology*, 65 (2019), 451-457.
Gindi, Renee, and Robin A Cohen, "Assessing measurement error in Medicare coverage from the National Health Interview Survey," *Medicare & Medicaid Research Review*, 2 (2012), mmrr. 002.002. a005.
Glied, S., and A. Lleras-Muney, "Technological innovation and inequality in health," *Demography*, 45 (2008), 741-761.
Goldin, Claudia, "America's graduation from high school: The evolution and spread of secondary schooling in the twentieth century," *Journal of Economic History*, 58 (1998), 345-374.
Goldin, Claudia, and Adriana Lleras-Muney, "XX> XY?: The changing female advantage in life expectancy," *Journal of Health Economics*, 67 (2019), 102224.
Goldin, J., I. Z. Lurie, and J. McCubbin, "Health Insurance and Mortality: Experimental Evidence from Taxpayer Outreach," *Quarterly Journal of Economics*, 136 (2021), 1-49.
Goldsmith-Pinkham, Paul, Maxim Pinkovskiy, and Jacob Wallace, "The great equalizer: Medicare and the geography of consumer financial strain," Working paper No. 31223. (Cambridge, MA: *National Bureau of Economic Research*, 2023).
Gompertz, Benjamin, "XXIV. On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. In a letter to Francis Baily, Esq. FRS &c," *Philosophical transactions of the Royal Society of London*, (1825), 513-583.
Goodman-Bacon, A., "Public Insurance and Mortality: Evidence from Medicaid Implementation," *Journal of Political Economy*, 126 (2018), 216-262.
---, "The Long-Run Effects of Childhood Insurance Coverage: Medicaid Implementation, Adult Health, and Labor Market Outcomes," *American Economic Review*, 111 (2021), 2550-2593.
Goodman-Bacon, Andrew J, and Sayeh S Nikpay, "Per capita caps in Medicaid—lessons from the past," *New England Journal of Medicine*, 376 (2017), 1005-1007.
Gornick, Marian E, Joan L Warren, Paul W Eggers, James D Lubitz, Nancy De Lew, Margaret H Davis, and Barbara S Cooper, "Thirty years of Medicare: impact on the covered population," *Health Care Financing Review*, 18 (1996), 179.
Graves, J. A., L. A. Hatfield, W. Blot, N. L. Keating, and J. M. McWilliams, "Medicaid Expansion Slowed Rates Of Health Decline For Low-Income Adults In Southern States," *Health Affairs*, 39 (2020), 67-76.
Grossman, Michael, "On the Concept of Health Capital and the Demand for Health," *Journal of Political Economy*, 80 (1972), 223-255.
Haider, Steven, and Gary Solon, "Life-cycle variation in the association between current and lifetime earnings," *American Economic Review*, 96 (2006), 1308-1320.
Handwerker, Elizabeth Weber, "What can the Social Security Notch tell us about the impact of additional income in retirement?," *Journal of Economic and Social Measurement*, 36 (2011), 71-92.
Harris, Richard, "Medicare: All Very Hegelian," *The New Yorker*, 42 (1966), 29.
Hoynes, Hilary, Diane Whitmore Schanzenbach, and Douglas Almond, "Long-Run Impacts of Childhood Access to the Safety Net," *American Economic Review*, 106 (2016), 903-934.
Huh, Jason, and Julian Reif, "Did Medicare Part D reduce mortality?," *Journal of Health Economics*, 53 (2017), 17-37.
Human Mortality Database – USA country page (https://www.mortality.org/Country/Country?cntr=USA,
Hypertension Detection Follow-up Program Cooperative Group, "Five-Year Findings of the Hypertension Detection and Follow-up Program: II. Mortality by Race-Sex and Age," *Journal of the American Medical Association*, 242 (1979), 2572-2577.
Kavalieratos, Dio, Jennifer Corbelli, DI Zhang, J Nicholas Dionne-Odom, Natalie C Ernecoff, Janel Hanmer, Zachariah P Hoydich, Dara Z Ikejiani, Michele Klein-Fedyshin, and Camilla Zimmermann,

"Association between palliative care and patient and caregiver outcomes: a systematic review and meta-analysis," *Journal of the American Medical Association*, 316 (2016), 2104-2114.
Keehan, Sean P, Andrew J Madison, John A Poisal, Gigi A Cuckler, Sheila D Smith, Andrea M Sisko, Jacqueline A Fiore, and Kathryn E Rennie, "National Health Expenditure Projections, 2024–33: Despite Insurance Coverage Declines, Health To Grow As Share Of GDP: Article features National Health Expenditure Projections, 2024-33," *Health Affairs*, 44 (2025), 776-787.
Keller, Elena, Jade E Newman, Andreas Ortmann, Louisa R Jorm, and Georgina M Chambers, "How much is a human life worth? A systematic review," *Value in Health*, 24 (2021), 1531-1541.
Khwaja, Ahmed, "Estimating willingness to pay for medicare using a dynamic life-cycle model of demand for health insurance," *Journal of Econometrics*, 156 (2010), 130-147.
Kitagawa, Evelyn M, and Philip M Hauser, *Differential mortality in the United States: A study in socioeconomic epidemiology* (Cambridge: Harvard University Press, 1973).
Kline, Patrick, and Christopher R Walters, "Evaluating public programs with close substitutes: The case of Head Start," *Quarterly Journal of Economics*, 131 (2016), 1795-1848.
Leinonen, Raija, Eino Heikkinen, and Marja Jylhä, "Predictors of decline in self-assessments of health among older people—A 5-year longitudinal study," *Social Science & Medicine*, 52 (2001), 1329-1341.
Levinsky, Michal, and Miriam Schiff, "Lifetime cumulative adversity and physical health deterioration in old age: Evidence from a fourteen-year longitudinal study," *Social Science & Medicine*, 289 (2021), 114407.
Lleras-Muney, A., "The relationship between education and adult mortality in the United States," *Review of Economic Studies*, 72 (2005), 189-221.
Lleras-Muney, Adriana, and Flavien Moreau, "A Unified Model of Cohort Mortality," *Demography*, 59 (2022), 2109-2134.
Lleras-Muney, Adriana, Joseph Price, and Dahai Yue, "The association between educational attainment and longevity using individual-level data from the 1940 census," *Journal of Health Economics*, 84 (2022), 102649.
Ludwig, Jens, and Douglas L Miller, "Does Head Start improve children's life chances? Evidence from a regression discontinuity design," *Quarterly Journal of Economics*, 122 (2007), 159-208.
McWilliams, J Michael, Ellen Meara, Alan M Zaslavsky, and John Z Ayanian, "Use of health services by previously uninsured Medicare beneficiaries," *New England Journal of Medicine*, 357 (2007), 143-153.
McWilliams, J Michael, Alan M Zaslavsky, Ellen Meara, and John Z Ayanian, "Impact of Medicare coverage on basic clinical services for previously uninsured adults," *Journal of the American Medical Association*, 290 (2003), 757-764.
McWilliams, J. M., A. M. Zaslavsky, E. Meara, and J. Z. Ayanian, "Health insurance coverage and mortality among the near-elderly," *Health Affairs*, 23 (2004), 223-233.
Miller, S., N. Johnson, and L. R. Wherry, "Medicaid and Mortality: New Evidence from Linked Survey and Administrative Data," *Quarterly Journal of Economics*, 136 (2021), 1783-1829.
Miller, Sarah, and Laura R Wherry, "The long-term effects of early life Medicaid coverage," *Journal of Human Resources*, 54 (2019), 785-824.
Moon, J Robin, Naoki Kondo, M Maria Glymour, and SV Subramanian, "Widowhood and mortality: a meta-analysis," *PloS one*, 6 (2011), e23465.
Moon, Marilyn, "What Medicare has meant to older Americans," *Health Care Financing Review*, 18 (1996), 49.
Multiple Risk Factor Intervention Trial Research Group, "Multiple Risk Factor Intervention Trial: Risk Factor Changes and Mortality Results," *Journal of the American Medical Association*, 248 (1982), 1465-1477.
---, "Mortality rates after 10.5 years for participants in the Multiple Risk Factor Intervention Trial. Findings related to a priori hypotheses of the trial," *Journal of the American Medical Association*, 263 (1990), 1795-1801.
---, "Mortality after 16 years for participants randomized to the Multiple Risk Factor Intervention Trial," *Circulation*, 94 (1996), 946-951.
National Center for Health Statistics, “Vital Statistics of the United States, 1960,” Working paper No. (Washington, D.C.: *U.S. Government Printing Office*, 1963).
Newson, Roger B, and Milena Falcaro, "Robit regression in Stata," *The Stata Journal*, 23 (2023), 658-682.
Niikura, Ryota, Yoshihiro Hirata, Nobumi Suzuki, Atsuo Yamada, Yoku Hayakawa, Hirobumi Suzuki, Shinzo Yamamoto, Ryo Nakata, Junko Komatsu, and Makoto Okamoto, "Colonoscopy reduces colorectal cancer mortality: A multicenter, long-term, colonoscopy-based cohort study," *PloS one*, 12 (2017), e0185294.
Noghanibehambari, Hamid, and Jason Fletcher, “In Money, We Survive: The Effects of Social Security Retirement Income on Longevity,” Working paper No. 34199. (Cambrige, MA: *National Bureau of Economic Research*, 2025).

Remarks with President Truman at the signing in Independence of the Medicare bill (https://www.presidency.ucsb.edu/documents/remarks-with-president-truman-the-signing-independence-the-medicare-bill,
KFF Survey of Consumer Experiences with Health Insurance (https://www.kff.org/affordable-care-act/kff-survey-of-consumer-experiences-with-health-insurance/, KFF (Kaiser Family Foundation)).
Polsky, D., J. A. Doshi, J. Escarce, W. Manning, S. M. Paddock, L. Cen, and J. Rogowski, "The health effects of Medicare for the near-elderly uninsured," *Health Services Research*, 44 (2009), 926-945.
Price, J., K. Buckles, J. Van Leeuwen, and I. Riley, "Combining family history and machine learning to link historical records: The Census Tree data set," *Explorations in Economic History*, 80 (2021), 101391.
Rambachan, Ashesh, and Jonathan Roth, "A more credible approach to parallel trends," *Review of Economic Studies*, 90 (2023), 2555-2591.
Ray, Kausik K, Sreenivasa Rao Kondapally Seshasai, Sebhat Erqou, Peter Sever, J Wouter Jukema, Ian Ford, and Naveed Sattar, "Statins and all-cause mortality in high-risk primary prevention: a meta-analysis of 11 randomized controlled trials involving 65 229 participants," *Archives of Internal Medicine*, 170 (2010), 1024-1031.
Rios-Avila, Fernando, Pedro Sant'Anna, and Brantly Callaway, "CSDID: Stata module for the estimation of Difference-in-Difference models with multiple time periods," (2023).
Roberts, Eric T, Jessica Phelan, Aaron L Schwartz, Ellen R Meara, Dominic Ruggiero, Lilly Estenson, Rachel M Werner, and José F Figueroa, "Loss of Subsidized Drug Coverage and Mortality Among Low-Income Medicare Beneficiaries," *New England Journal of Medicine*, 392 (2025).
Robins, James, "A new approach to causal inference in mortality studies with a sustained exposure period—application to control of the healthy worker survivor effect," *Mathematical modelling*, 7 (1986), 1393-1512.
Roth, Jonathan, and Pedro H. C. Sant'Anna, "When Is Parallel Trends Sensitive to Functional Form?," *Econometrica*, 91 (2023), 737-747.
Rubin, Donald B, "Estimating causal effects of treatments in randomized and nonrandomized studies," *Journal of Educational Psychology*, 66 (1974), 688.
Ruggles, Steven, Matt A Nelson, Matthew Sobek, Catherine A Fitch, Ronald Goeken, J David Hacker, Evan Roberts, and J Robert Warren, "IPUMS Ancestry Full Count Data: Version 4.0 [dataset]. Minneapolis, MN: IPUMS," (Minneapolis, MN: IPUMS, 2024).
Rust, John, and Christopher Phelan, "How Social Security and Medicare Affect Retirement Behavior In a World of Incomplete Markets," *Econometrica*, 65 (1997), 781-831.
Schwandt, Hannes, "Wealth shocks and health outcomes: Evidence from stock market fluctuations," *American Economic Journal: Applied Economics*, 10 (2018), 349-377.
Seaman, Shaun R, and Ian R White, "Review of inverse probability weighting for dealing with missing data," *Statistical Methods in Medical Research*, 22 (2013), 278-295.
Smith, David Barton, *The Power to Heal: Civil Rights, Medicare, and the Struggle to Transform America's Health Care System* (Vanderbilt University Press, 2016).
Snyder, Stephen E, and William N Evans, "The effect of income on mortality: evidence from the social security notch," *Review of Economics and Statistics*, 88 (2006), 482-495.
Cohort Life Tables (https://www.ssa.gov/oact/HistEst/CohLifeTables/2020/CohLifeTables2020.html, Office of the Chief Actuary).
Starr, P., *The Social Transformation of American Medicine: The Rise Of A Sovereign Profession And The Making Of A Vast Industry* (Basic Books, 1982).
Stevens, Rosemary A, *In Sickness and In Wealth: American Hospitals in the 20th Century* (London: Johns Hopkins University Press, 1989).
---, "Health care in the early 1960s," *Health Care Financing Review*, 18 (1996), 11.
Thomasson, Melissa A, "The importance of group coverage: How tax policy shaped US health insurance," *American Economic Review*, 93 (2003), 1373-1384.
Thomasson, Melissa A., "Early evidence of an adverse selection death spiral? The case of Blue Cross and Blue Shield," *Explorations in Economic History*, 41 (2004), 313-328.
Thompson, Owen, "The long-term health impacts of Medicaid and CHIP," *Journal of Health Economics*, 51 (2017), 26-40.
U.S. Census Bureau., "Survey of Income and Education, 1976," (Inter-university Consortium for Political and Social Research [distributor], 2006).
US Census Bureau, "Survey of Income and Education, 1976: Welfare Extract," (Inter-university Consortium for Political and Social Research [distributor], 1992).
Van Tiggelen, R, and E Pouders, "Ultrasound and computed tomography: Spin-offs of the world wars," *JBR-BTR*, 86 (2003), 235-241.
West, Howard, "Five years of Medicare-A statistical review," *Social Security Bulletin*, 34 (1971), 17.

Wherry, L. R., and B. D. Meyer, "Saving Teens Using a Policy Discontinuity to Estimate the Effects of Medicaid Eligibility," *Journal of Human Resources*, 51 (2016), 556-588.
Wherry, L. R., S. Miller, R. Kaestner, and B. D. Meyer, "Childhood Medicaid Coverage and Later-Life Health Care Utilization," *Review of Economics and Statistics*, 100 (2018), 287-302.
Wright, Janice C., and Milton C. Weinstein, "Gains in Life Expectancy from Medical Interventions — Standardizing Data on Outcomes," *New England Journal of Medicine*, 339 (1998), 380-386.
Wyse, Angela, and Bruce D Meyer, "Saved by Medicaid: new evidence on health insurance and mortality from the universe of low-income adults," Working paper No. 33719 (Cambridge, MA: *National Bureau of Economic Research*, 2025).
Yue, Dahai, Susan L Ettner, Jack Needleman, and Ninez A Ponce, "Selective mortality and nonresponse in the Health and Retirement Study: implications for health services and policy research," *Health Services and Outcomes Research Methodology*, 23 (2023), 313-336.
Yue, Dahai, Ninez A. Ponce, Jack Needleman, Susan L. Ettner, and Adriana Lleras-Muney, "Increased Schooling Reduces Hospitalization Later in Life: New Evidence with Optimal Instruments from the United States," *American Journal of Health Economics*, 3 (2023), 375-418.

Table 1: Characteristics of white individuals linked to the 1940 full-count census

	20% Census-Tree data (N=3,697,163)		1940 full-count census (N=57,416,201)
	w/o IPW	w/ IPW	Percent or Mean
Birth cohorts based on 1940 census			
1883-1884	0.12%	0.40%	3.76%
1885-1889	10.56%	11.15%	10.91%
1890-1894	15.00%	13.72%	13.09%
1895-1899	15.85%	14.23%	13.51%
1900-1904	17.29%	15.89%	14.95%
1905-1909	17.90%	17.13%	15.75%
1910-1914	20.95%	23.15%	20.91%
1915-1917	2.32%	4.32%	7.12%
Sex			
Male	53.69%	50.38%	50.21%
Female	46.31%	49.62%	49.79%
Hispanics			
Not Hispanic	99.36%	98.73%	98.49%
Mexican	0.45%	0.94%	1.07%
Puerto Rican	0.01%	0.04%	0.09%
Cuban	0.01%	0.03%	0.04%
Other	0.17%	0.26%	0.31%
Regions of residence in 1940			
Northeast	17.80%	28.09%	30.50%
Midwest	37.25%	33.86%	32.74%
South	30.94%	25.92%	25.01%
West	14.01%	12.13%	11.74%
Regions of birth			
Northeast	16.87%	24.42%	24.27%
Midwest	39.42%	33.54%	31.86%
South	32.30%	26.83%	25.69%
West	6.72%	5.36%	5.06%
US or outlying areas	0.02%	0.06%	0.12%
Abroad	4.67%	9.80%	13.00%
Marital status			
Married, spouse present	84.19%	75.35%	72.29%
Married, spouse absent	1.81%	3.18%	3.75%
Separated	0.00%	0.00%	0.00%
Divorced	1.16%	1.82%	1.87%
Widowed	2.04%	3.21%	3.94%
Never married/single	10.80%	16.44%	18.14%
Veteran status			
N/A	97.30%	97.46%	97.49%
Not a veteran	1.14%	1.09%	1.11%
Veteran	0.32%	0.28%	0.25%
Unknown	1.24%	1.17%	1.15%
Educational level			
0-7	22.25%	22.29%	23.59%
8-11	45.94%	44.62%	44.45%
12+	29.89%	31.01%	29.60%
Missing	1.91%	2.08%	2.36%
Years of schooling (SE)	9.35 (3.25)	9.33 (3.40)	9.16 (3.46)
Household total annual income in 1940 US dollars (SE)	1295.45 (1876.93)	1531.80 (2687.66)	1533.34 (2491.17)
Number of own family members in household (SE)	4.34 (2.05)	3.92 (2.03)	3.82 (2.06)
Occupational income score in 1940 (SE)	14.96 (14.59)	15.16 (14.56)	14.99 (14.40)

Notes: The inverse probability weights (IPW) are estimated using a robit model that predicts a match as a function of the following as covariates: female dummy, years of schooling, age, 6 marital status dummies, number of children, labor force participation dummy, occupation dummies, occupation income scores, wage and salary income, veteran status (yes, no, unknown), place of birth, and state of residence in 1940. We estimated the model separately for men and women.

Table 2: Estimates of Medicare's average treatment effect on life expectancy at age 65 overall, by education, by income in 1940, and by cohort

	Sample Size	Life expectancy at age 65	ITS	DID
Panel A. Men				
All Men	1,985,048	14.43	0.87	1.20
			(0.72, 1.00)	(0.91, 1.49)
Years of Schooling				
0-7	465,407	13.60	1.02	1.18
			(0.79, 1.26)	(0.78, 1.65)
8-11	922,526	14.14	0.86	0.99
			(0.66, 1.05)	(0.64, 1.37)
12+	557,896	15.43	0.67	0.85
			(0.38, 1.03)	(0.20, 1.56)
1940 Household Income Tertiles				
Low income	661,949	14.39	0.67	1.25
			(0.42, 0.92)	(0.86, 1.77)
Middle income	661,470	14.19	1.03	1.29
			(0.77, 1.25)	(0.79, 1.86)
High income	661,629	14.67	0.85	1.07
			(0.60, 1.09)	(0.62, 1.56)
Cohort Groups				
1885-1890	314,523	13.92	0.29	0.30
			(0.22, 0.38)	(-0.04, 0.69)
1891-1900	638,480	13.94	0.64	0.86
			(0.50, 0.79)	(0.70, 1.03)
1901-1908	552,895	14.38	1.48	1.54
			(1.21, 1.75)	(1.12, 1.97)
1909-1915	479,150	15.06	0.62	1.37
			(0.30, 0.95)	(0.92, 1.87)
Panel B. Women				
All Women	1,712,115	18.83	-0.80	0.87
			(-1.11, -0.52)	(0.35, 1.39)
Years of Schooling				
0-7	357,273	17.49	0.21	0.80
			(-0.34, 0.69)	(0.12, 1.55)
8-11	776,058	18.63	-0.75	0.81
			(-1.23, -0.37)	(0.16, 1.45)
12+	547,351	19.81	-1.61	1.30
			(-2.31, -1.10)	(0.15, 2.47)
1940 Household Income Tertiles				
Low income	572,842	18.69	-0.78	0.49
			(-1.33, -0.35)	(-0.28, 1.30)
Middle income	575,573	18.61	-0.26	1.37
			(-0.75, 0.20)	(0.51, 2.23)
High income	563,700	19.12	-1.29	0.69
			(-1.88, -0.86)	(-0.15, 1.45)
Cohort Groups				
1885-1890	256,629	17.33	-0.06	0.33
			(-0.24, 0.12)	(-0.32, 0.99)
1891-1900	537,968	18.42	-0.60	0.24
			(-0.96, -0.31)	(-0.06, 0.53)
1901-1908	494,735	19.18	-0.85	1.27
			(-1.28, -0.42)	(0.54, 2.02)
1909-1915	422,783	19.39	-1.13	1.21
			(-1.69, -0.64)	(0.52, 1.93)

Notes: This table reports the estimated life expectancy gains associated with gaining access to Medicare. The Appendix contains the details of our computations. All estimates are produced using the Census Tree data. In parentheses are bias-corrected 95% confidence intervals produced by bootstrapping with 1,000 resamples. Mean Life expectancy at age 65 denotes weighted average cohort-specific life expectancy at age 65 using the number of people survived to Medicare eligibility age as weights.

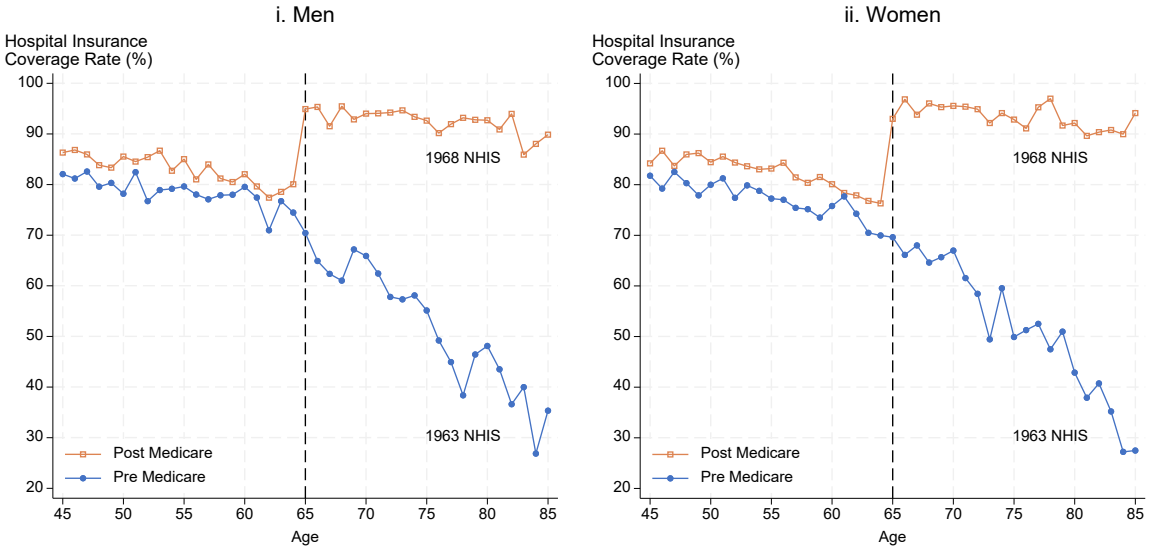
Table 3: Structural Model Mechanisms and Counterfactuals Using Men 1900-1909

	Parameter before Medicare	Parameter after Medicare	% change	Average MSE (Survival)	Implied gain in LE65 from matching RF at age 65	Predicted gain in LE55 from providing Medicare at age 55	Predicted loss in LE65 from providing Medicare at age 75
Panel A: Matching DiD endpoint, 5 years							
I	0.138	0.151	9.42%	0.0514	0.574	0.48	-0.37
δ	0.00096	0.00093	-3.13%	0.0372	0.595	0.37	-0.36
α	1.386	1.377	-0.65%	0.0472	0.654	0.38	-0.39
Panel B: Matching DiD endpoint, 10 years							
I	0.138	0.173	25.36%	3.7547	1.701	1.4	-1.12
δ	0.00096	0.00087	-9.38%	3.0362	1.76	1.08	-1.07
α	1.386	1.363	-1.66%	2.8942	1.755	1.02	-1.05

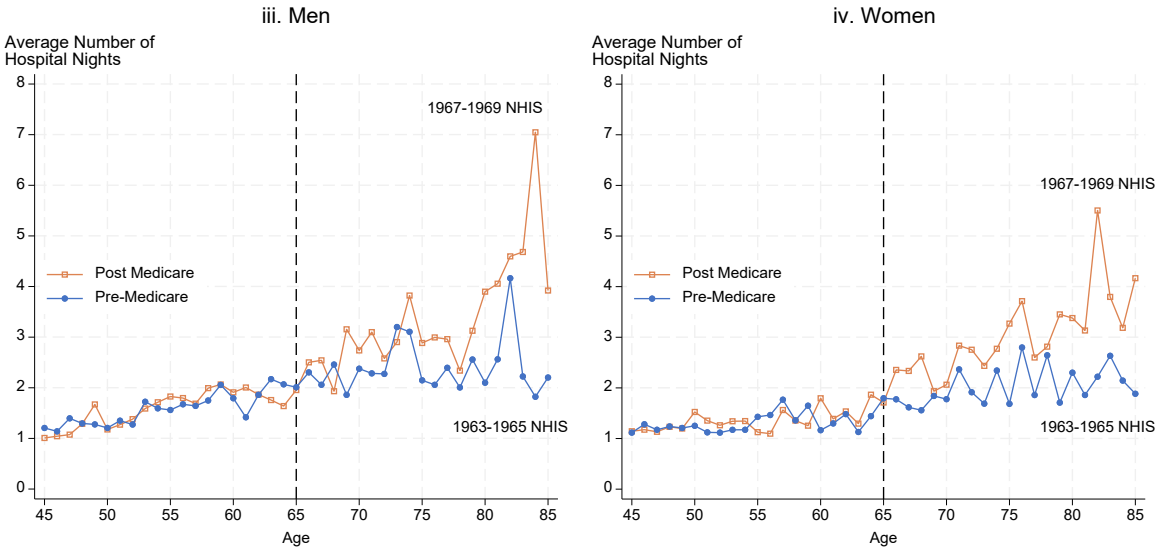
Notes: these estimates use our (pooled) event-study estimates for the 1900-1909 male cohorts as the reduced form estimates. Column (1) reports the average value of the model parameter when calibrating the model up to age 65. Column (2) reports the average value of the parameter when it is allowed to vary to match the reduced form effect of Medicare. Column (3) reports the percent change from (1) to (2). Column (4) reports fit on the survival curve. Column (5) reports the estimated effect of Medicare on LE65 in the model. This is computed by comparing the LE with the change in the parameters at age 65 with the prediction of the model when the parameter is allowed to change at age 65. Column (6) reports the marginal gain to LE55 from providing Medicare at age 55, where the predicted change in LE55 is computed by comparing a model where the relevant parameter is changed at age 65 to match the reduced form treatment effect to a model where the same parameter change occurs at age 55. Column (7) reports the marginal loss to LE65 of delaying Medicare eligibility to age 75 in an equivalent exercise.

Figure 1: Hospital insurance coverage and hospital nights before and after Medicare's introduction

a. Health Insurance Coverage



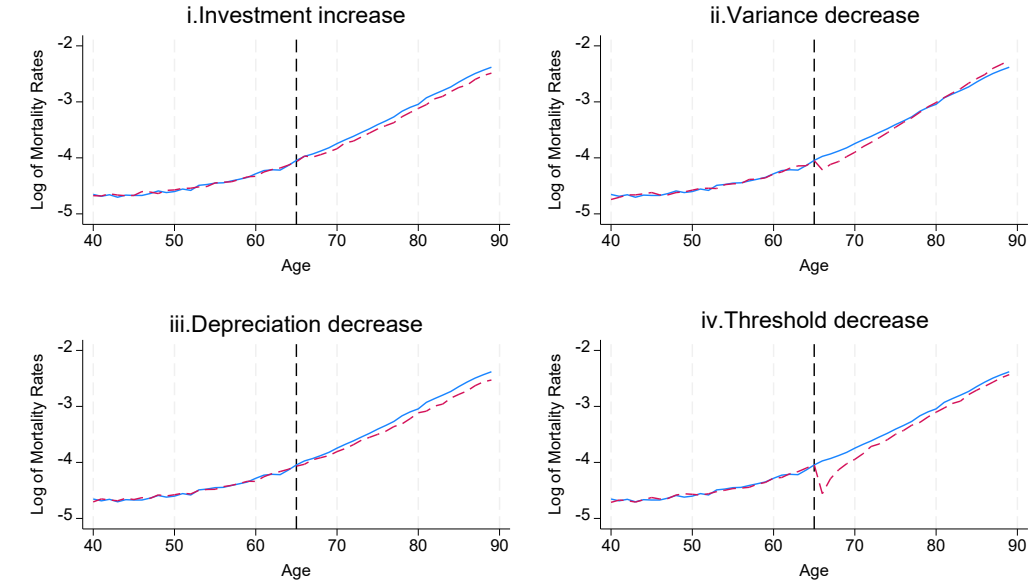
b. Hospital Nights



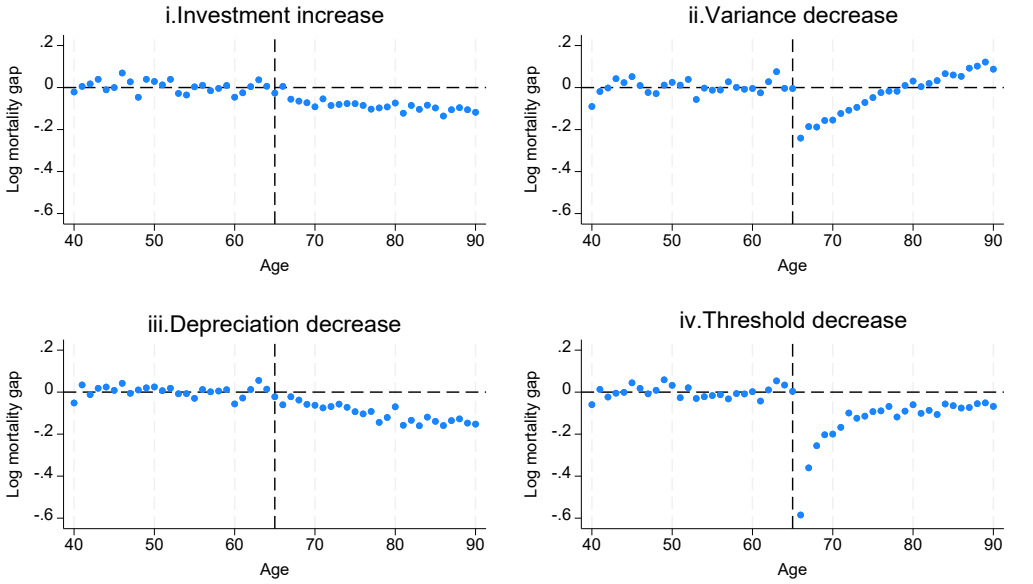
Notes: The figure shows hospital insurance rate and the average number of nights in hospital in the past 12 months for whites by sex. We use data from the National Health Interview Survey (NHIS), a nationally representative survey of the noninstitutionalized population. Specifically, we use the 1963 and 1968 NHIS that ask if one individual had hospital insurance coverage for Panels A and B. The number of nights in hospital in the past 12 months was asked in 1963-1969 NHIS. We use the 1963-1969 NHIS data from IPUMS that cleaned the hospital nights variable for Panels C and D; we exclude 1966 in the analysis because Medicare was introduced in the middle of 1966. Survey weights are applied to make the estimates representative of the U.S. population.

Figure 2: Simulated changes of Gompertz curves after permanent unanticipated shocks at age 65

a. log of mortality with and without parameter changes at age 65

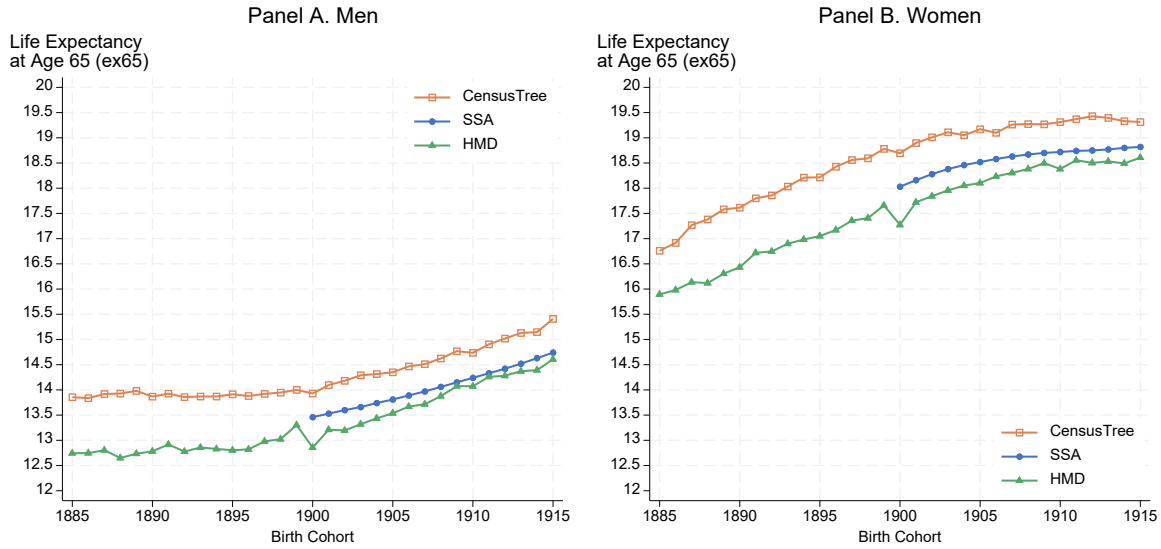


b. Effects of parameter changes at age 65 on log mortality



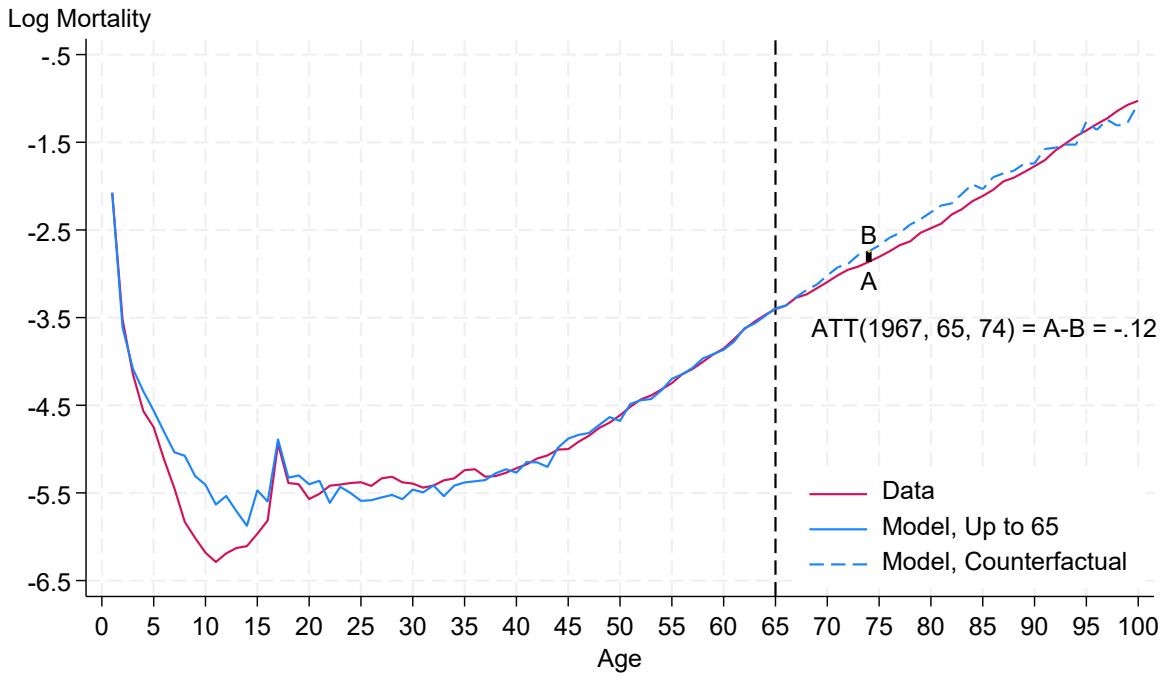
Notes: The figures show simulated results based on Lleras-Muney and Moreau (2022)'s unified model for cohort mortality, with parameter values for the baseline model given by $I = 0.4$, $\delta = 0.0006$, $\sigma = 1$, $\alpha = 1.7$, $\mu_0 = 0.9$, and an adolescent accident shock $\kappa = 0.008$. The threshold for dying is set at 0. In the top panel, the solid blue line shows the evolution of (the natural logarithm of) mortality rates by age for the baseline population and the dashed red line shows shifted Gompertz curves after permanent shocks are simulated at age 65: I is increased by 10% (investment increase), the variance σ is decreased to 0.6 (variance decrease), accidents are decreased to 0.004 (accident decrease), the depreciation rate δ is lowered by 5% (depreciation decrease), and finally the threshold is lowered from 0 to -1 (threshold decrease). In the bottom panel (panel b) we show the implied evolution of the gaps in (natural) logs, which are given by the distance between the solid blue line and the dashed red line in panel a.

Figure 3: The expected age at death conditional on survival to age 65.



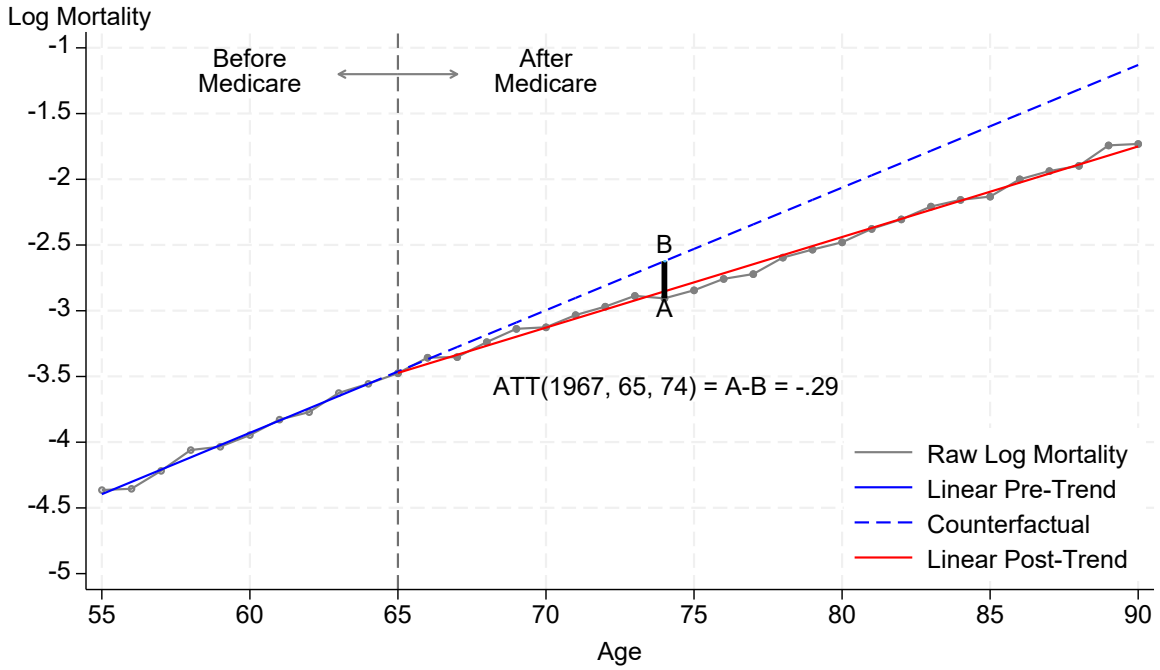
Notes: Shown are life expectancy at age 65 ($ex65$) for men and women. The orange hollow squares represent $ex65$ constructed from a 20% random sample of whites born 1885-1915 from our Census Tree data. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. The blue circles denote $ex65$ from the Social Security Administration (SSA) cohort life tables. The SSA, available only for cohorts born in 1900 or later, include all races and are not disaggregated by race. The green triangles are $ex65$ computed based on the U.S. cohort death rates for all races from the Human Mortality Database (HMD).

Figure 4: Identification of Medicare's effect on one cohort's log mortality rates in the structural design - men born in 1902



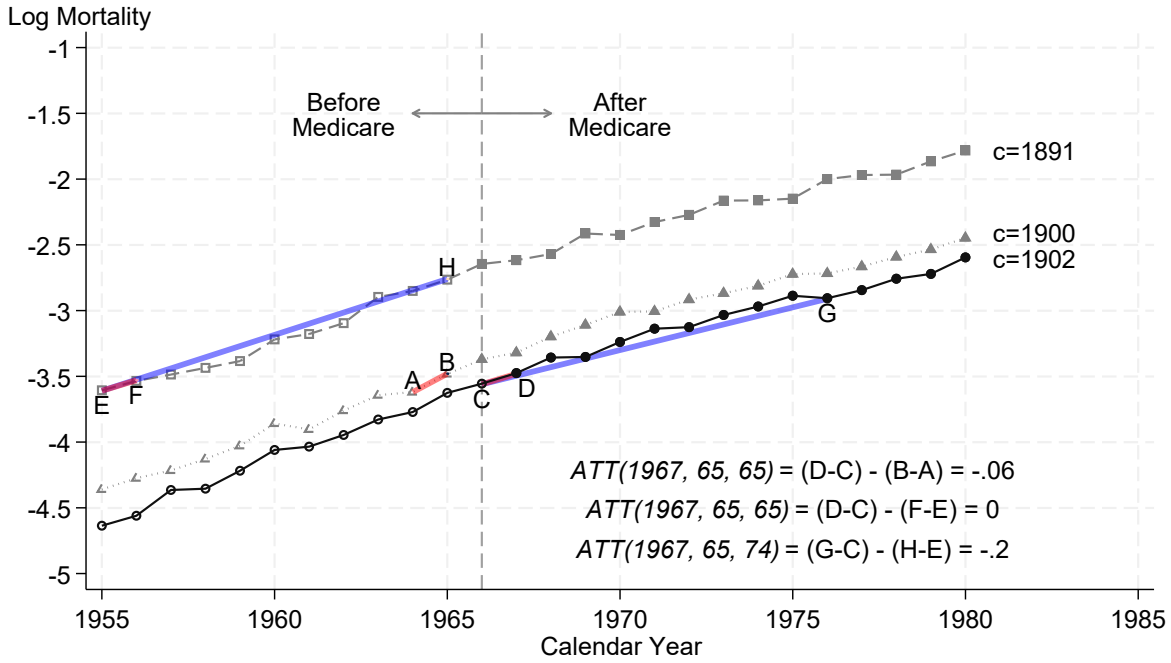
Notes: This figure demonstrate how the model can be used for identification using the Social Security Administration cohort tables starting at birth. The y-axis shows the natural log of mortality. The blue line shows these rates for the 1902 cohort of men in the Social Security Administration data. The model estimates are produced in two steps. We first estimate the model using data up to age 65. We then project mortality rates beyond age 65 using the estimated parameters (solid red line). The estimated parameters for this cohort and other cohorts we study are presented in Table A2.

Figure 5: Identification of Medicare's effect on one cohort's log mortality rates in the interrupted time series design - men born in 1902



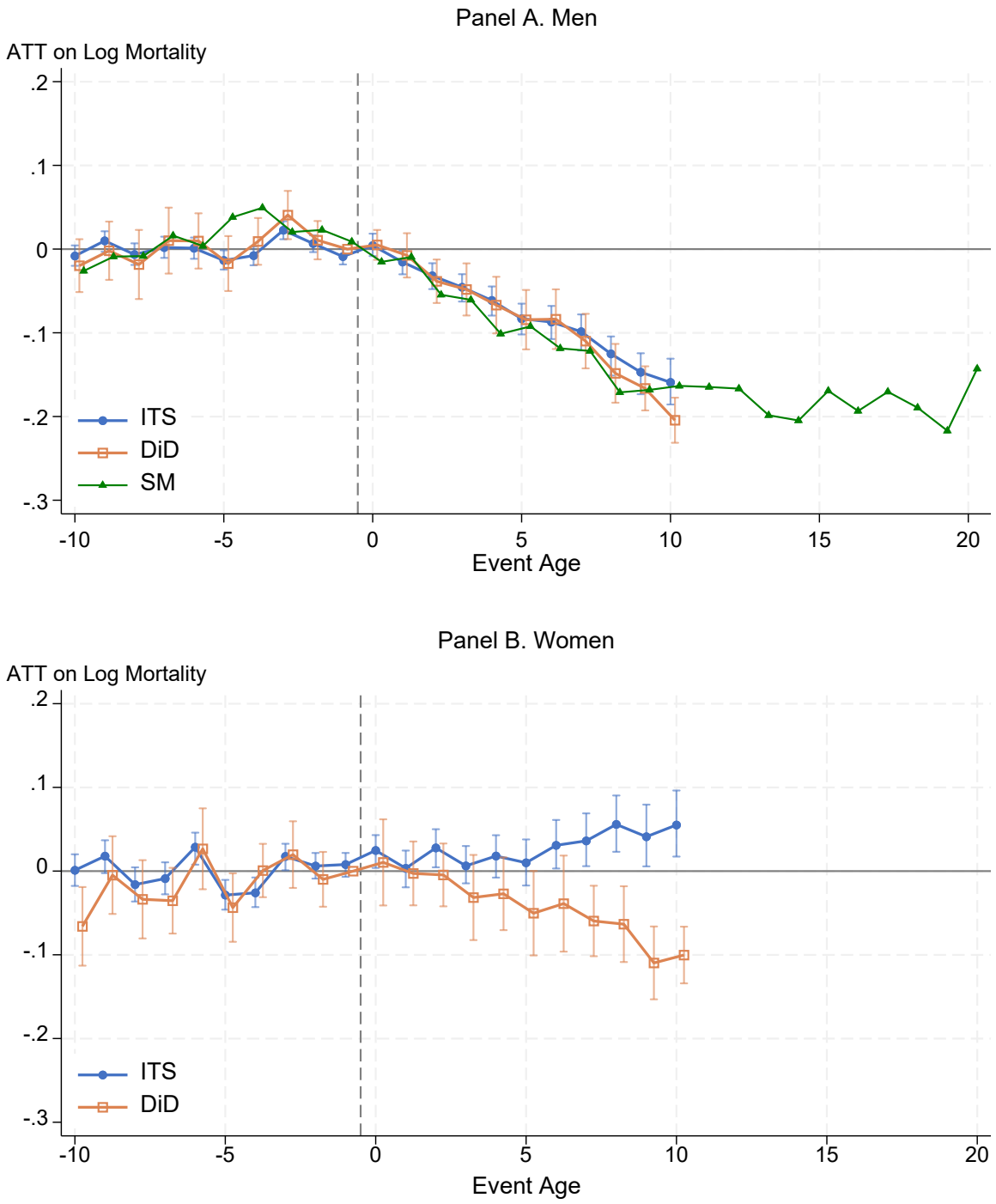
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. The figures show the log mortality (Gompertz curve) for the 1902 cohort for white men. The blue solid line is a linear fit of log mortality against age over 10 years prior to Medicare eligibility age (pre-trend). The blue dashed line is a linear project of the pre-trend of log mortality after Medicare eligibility to age 90, representing the counterfactual without Medicare's introduction. The red solid line represents the linear fit of the post-Medicare trend in log mortality from Medicare eligibility age to age 90.

Figure 6: Identification of Medicare's effect on one cohort's log mortality rates in the difference-in-differences design - men born in 1902



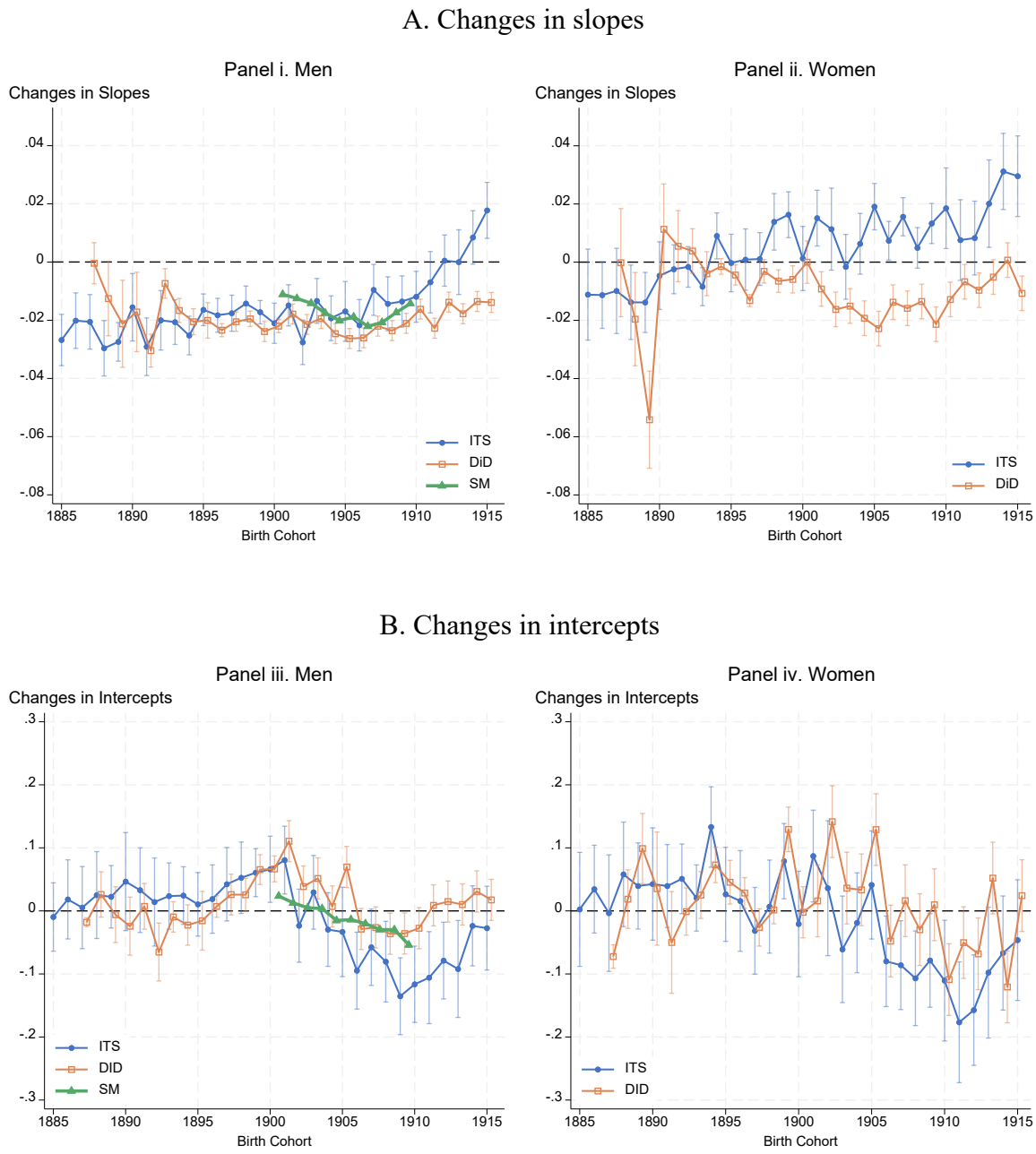
Notes: This figure highlights the estimator for $ATT(g_t, g_a, a)$ for the 1902 men cohort as an example. We describe the treatment effect for the Medicare eligibility year ($g_t = 1967$) and 9 years after the introduction. This effect is identified by comparing the change in the (log) mortality rate before (C) and after becoming eligible for Medicare (D) (at ages 64 and 65) for the 1902 cohort, to the change for each of the 1891-1900 cohorts in the previous year prior to becoming eligible for Medicare; note as explained in the text, we restricted the comparisons to nearby cohorts. The identifying assumption is parallel trends between pairs of cohorts and pairs of ages ($PT(c, a)$), namely that the change in mortality for the 1891-1900 cohort between ages 64 and 65 (e.g., B-A, or F-E) serves as a counterfactual for the change in mortality for the 1902 cohort because they would have evolved in parallel if the 1902 cohort had not been treated. This assumption can be partially tested by checking whether the mortality rates of the two cohorts were in fact parallel prior to age 65. Similarly, the treatment effect for the 1902 cohort 9 years after Medicare use (and only use) the 1891 cohort as the comparison group. The implied estimate of the effect of Medicare 9 years after its introduction year for the 1902 cohort is illustrated in this figure as (G-C)-(H-E), which -0.2 for men.

Figure 7: Estimates of Medicare's average treatment effect on log mortality rates by event-age



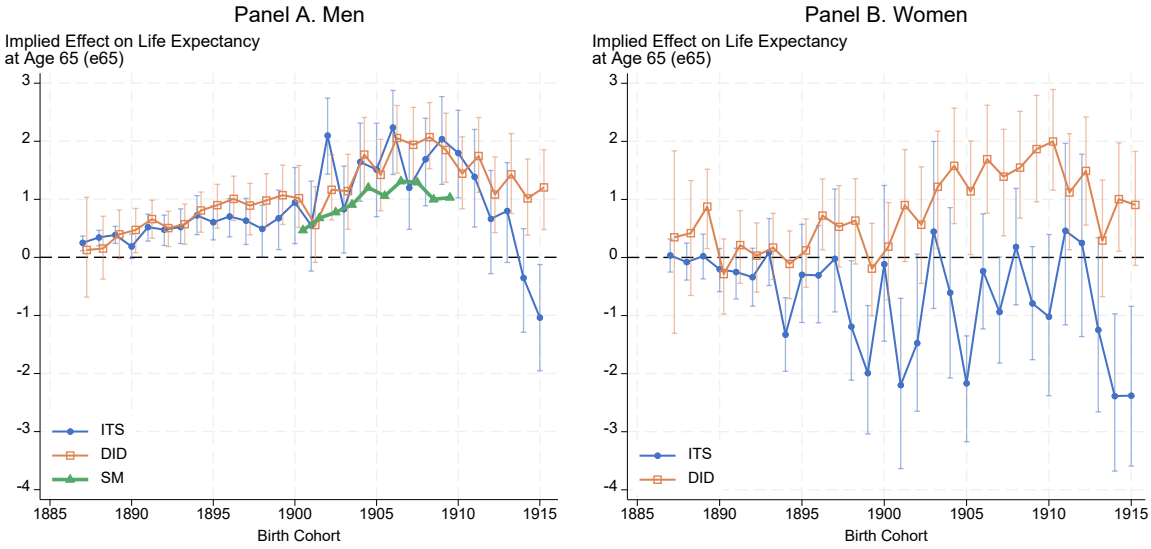
Notes: The figure plots the estimates of $ATT(e)$ parameters from equation (3) for men and women using both the ITS and DiD designs using a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. The blue circles, orange squares, and green triangles represent event-study estimates from the interrupted time series (ITS), difference-in-differences (DiD), and structural model (SM), respectively. The green triangle estimates are derived from the Social Security Administration (SSA) cohort life tables, which are available only for the 1900 and later cohorts. The $ATT(e)$ estimates from the SM design are only computed from men born 1900-1909, for which our structural model provides a good fit using the SSA life tables.

Figure 8: Estimates of Medicare's average treatment effect on the intercept and slope of log mortality rates by cohort



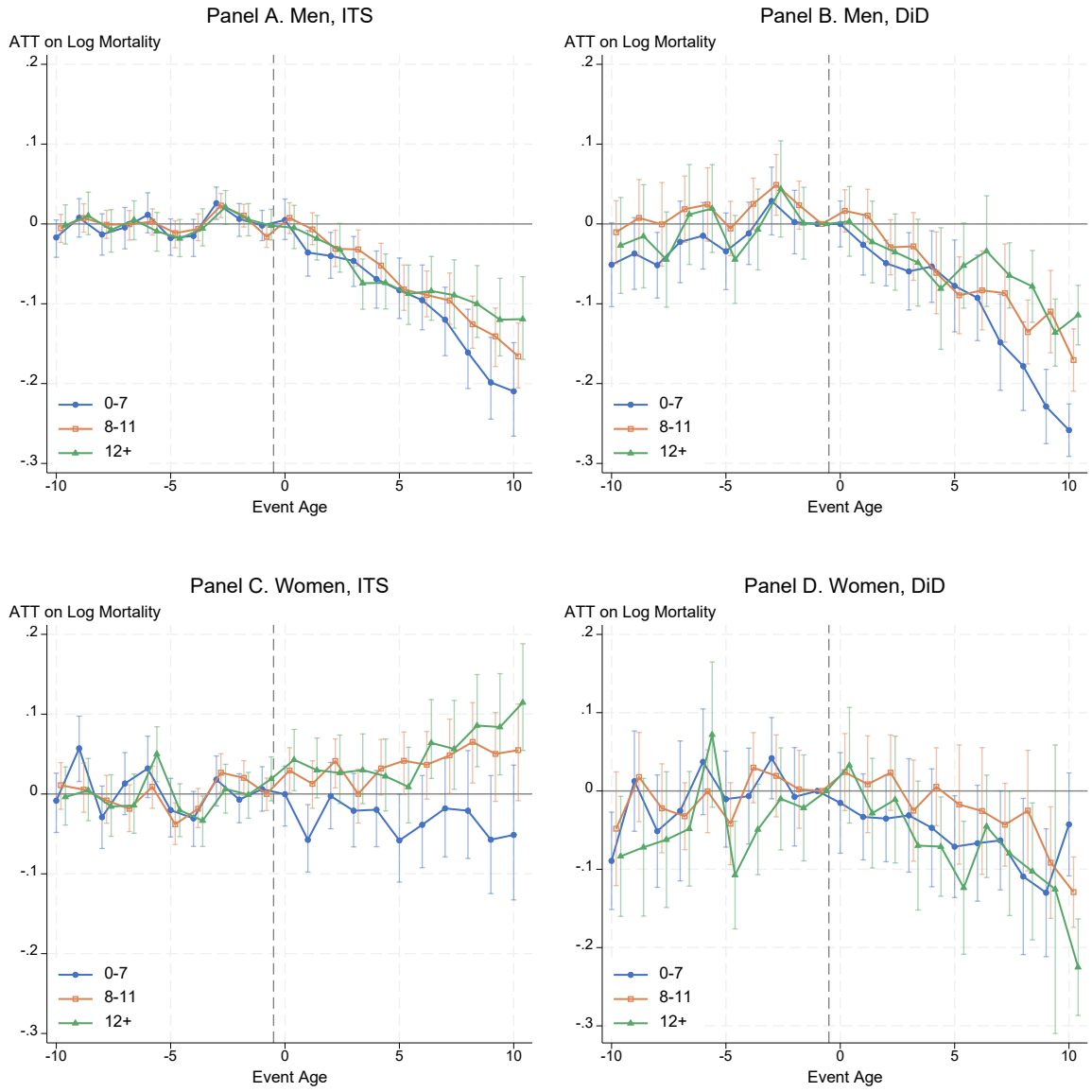
Notes: We use three approaches to estimate changes in slopes and levels of log mortality, after Medicare started in 1966, by sex and birth cohort using three different methods. The blue circle (ITS approach) and orange square (DiD approach) estimates are from a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. The green triangle estimates are derived from the Social Security Administration (SSA) cohort life tables, which are available only for the 1900 and later cohorts. We compute the slope and intercept estimates only for men born 1900-1909, for which our structural model provides a good fit using the SSA life tables.

Figure 9: Estimates of Medicare's average treatment effect on life expectancy at age 65 by cohort



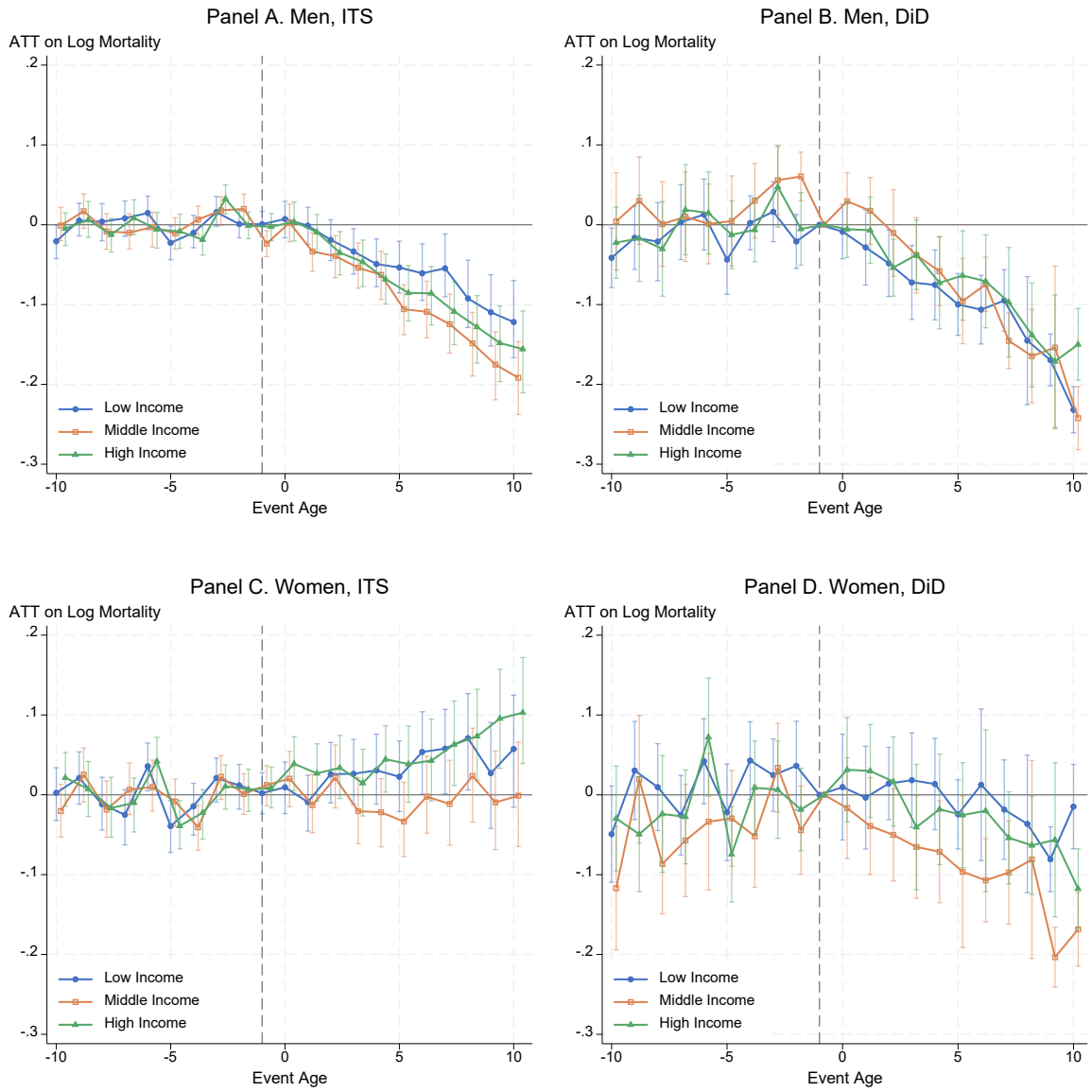
Notes: Shown are the implied effects of Medicare on life expectancy at age 65 based on our ITS, DiD and structural model (SM) estimation approaches by sex, and birth cohort. The blue (circle) and orange (square) estimates are from a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. We use a bootstrapping approach with 1,000 resamples to obtain the 95% confidence intervals for our analysis using Census Tree microdata. The green triangle estimates correspond to the structural model estimates the Social Security Administration (SSA) cohort life tables, which are available only for the 1900 and later cohorts. The SM estimates are only estimated for men born 1900-1909, for which our structural model provides a good fit using the SSA life tables.

Figure 10: Estimates of Medicare's average treatment effect on log mortality rates by event-age and education group



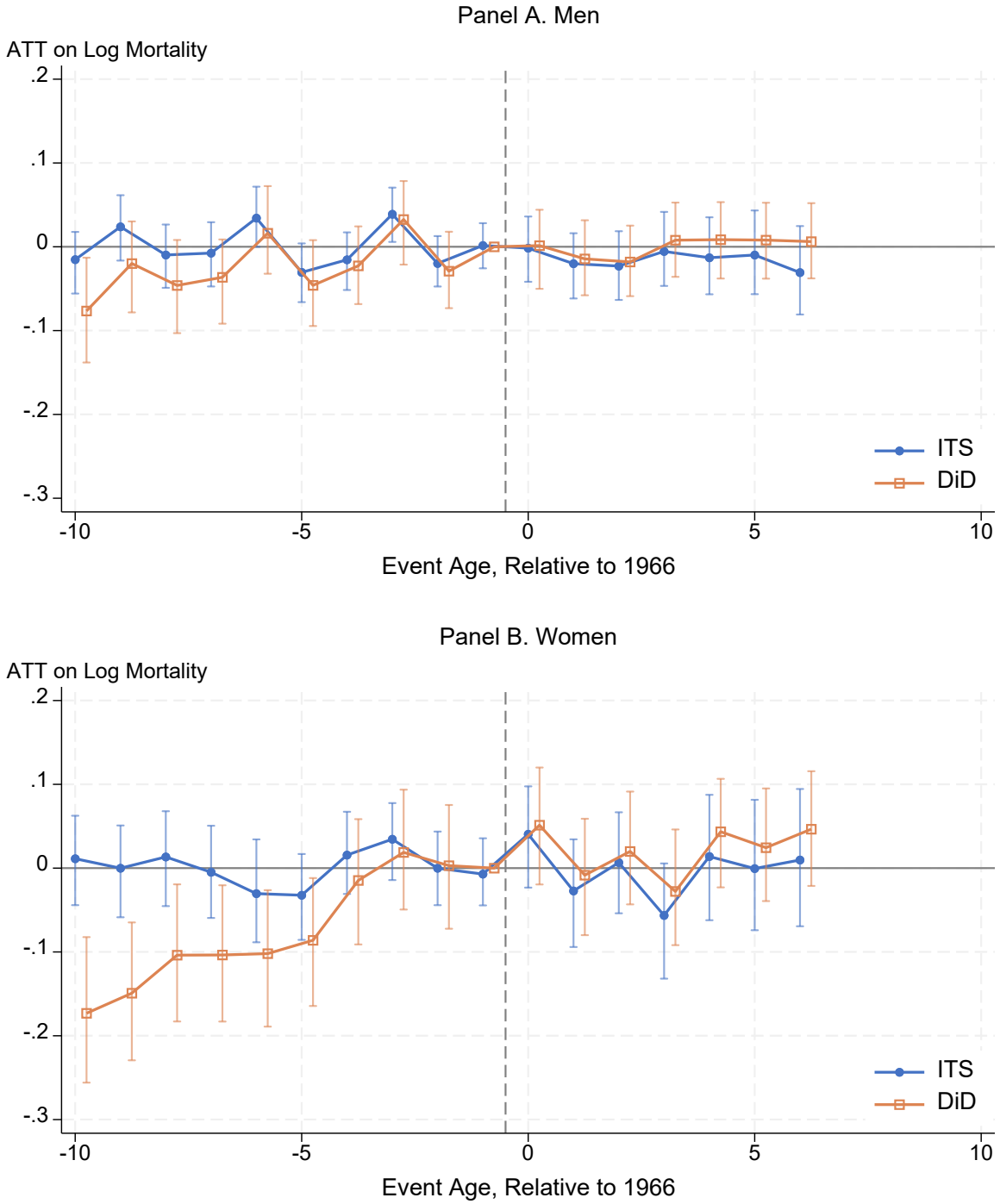
Notes: Shown are estimated event-study estimates from the ITS and DiD approaches, stratified by sex and educational group. We use a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. We use a bootstrapping approach with 1,000 resamples to obtain the 95% confidence intervals for the ITS event-study estimates using Census Tree microdata. We obtain years of schooling from the 1940 census and categorize respondents into four groups: 0-7 years of schooling, 8-11 years of schooling, 12 or more years of schooling, and missing years of schooling.

Figure 11: Estimates of Medicare's average treatment effect on log mortality rates by event-age and income group



Notes: Shown are estimated event-study estimates from the ITS and DiD approaches, stratified by sex and income group. We use a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. We use a bootstrapping approach with 1,000 resamples to obtain the 95% confidence intervals for the ITS event-study estimates using Census Tree microdata. We obtain household income from the 1940 census and categorize respondents into three income tertiles by sex: low income, middle income, and high income.

Figure 12: Estimates of Medicare's spillover effects and time shocks in 1966 for 1908-1915 cohorts



Notes: In this falsification test, we use aggregated log mortality data by sex and cohort but restrict to the pre-Medicare ages. We use 1966 as the treatment year and focus on the 1908-1915 cohorts who were aged 51-58 in 1966 and whom we can observe for at least 7 years prior to their gaining Medicare themselves. The figures show event study estimates from a DiD and ITS designs described in Appendix Section 6.